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JOURNAL

OF THE

Indian Medical Association

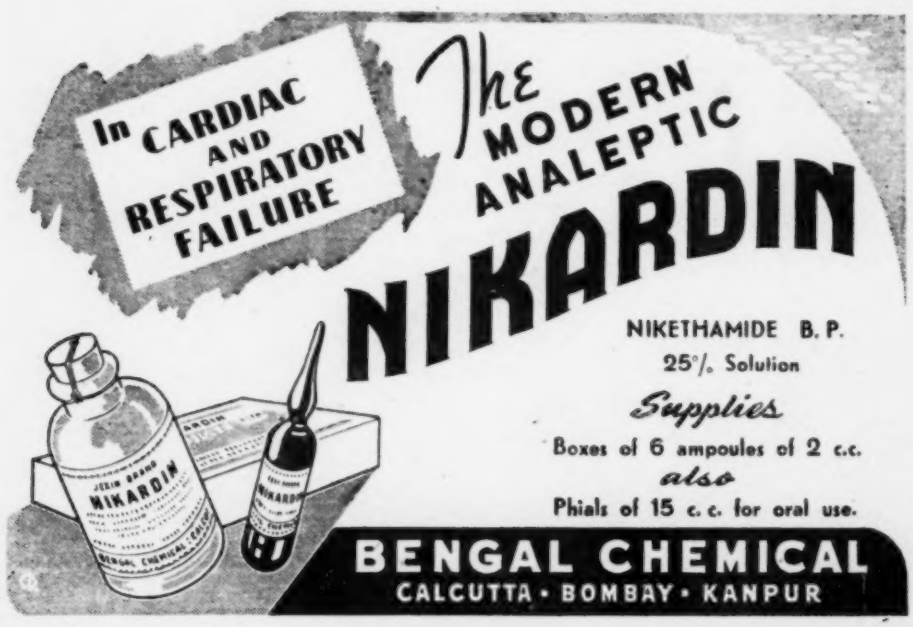
VOLUME XXI
NUMBER 3

CALCUTTA
DECEMBER 1951

Indian Medical Association
Central Office,
"Hanging Bridge", Daryaganj, Delhi-7

Office of the Journal
23, Samavaya Mansions,
Corporation Place, Calcutta-13

Annual Subscription
Inland Rs. 18/-
Foreign £1 10s. or $7



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SUPPLEMENT

Journal of the Indian Medical Association

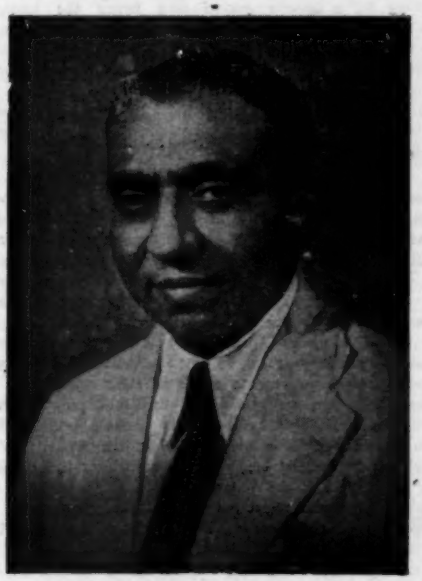
VOL. XXI, No. 3 CALCUTTA DECEMBER, 1951

THE FIFTH GUJARAT AND SAURASHTRA MEDICAL CONFERENCE

Under the auspices of Gujarat and Saurashtra provincial branch of the I.M.A., the fifth annual conference of medical men of Gujarat and Saurashtra was held at Nadiad in the Town Hall between 30th March and 1st April, 1951.

Dr. H. C. Aldrich, welcomed the guests on behalf of the Reception Committee and requested Dr. C. S. Patel, President, Medical Council of India, to inaugurate the Conference. Dr. C. S. Patel in his speech touched several important points on the question of medical education. After he had declared the conference open, Dr. Motibhai D. Patel, was requested to take the Presidential chair by the outgoing President, Dr. M. K. Dixit.

Dr. M. D. Patel took the chair amidst loud cheers. After the Presidential address the Chairman, Reception Committee,



DR. M. D. PATEL,
President, Gujarat and Saurashtra Provincial Conference.

requested the Municipal President to declare the Scientific and Pharmaceutical Exhibition open. Lastly, the subjects committee was formed and the house adjourned or lunch. The scientific session commenced in the afternoon the same day. It was declared open by Dr. G. B. Mankad of Ahmedabad. After the first scientific session was over the house adjourned at 6 P.M. A civic reception was arranged by the Nadiad Municipality. Interesting features were the Garden Party and a variety entertainment at 10 P.M. on the first day.

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The following are excerpts from the presidential address delivered by Dr. M. D. Patel at the Nadiad Conference:—

We are very thankful to the members of the Nadiad Branch for having invited us to hold the conference this year at Nadiad, a town renowned for Gujarati poets and authors. Nadiad again—I must admit—is made famous by that old American missionary doctor—Dr. Corporon, whom I had seen in actual work. About forty years ago first Dr. Gevin at Anand and later Dr. Corporon at Nadiad rendered great service to this part of Gujarat by giving medical relief, which is now being continued by Dr. Cook at Anand and Dr. Aldrich at Nadiad. The most important thing to my mind is that these pioneer doctors made people surgical-minded. The so-called ignorant vaidyas and such others with vested interests frightened the people of surgery. The missionary doctors came with new light and equipments of modern science and with great zeal to help humanity in their sufferings. The people soon realised the benefits of modern surgery from the results achieved by those doctors.

MEDICAL EDUCATION AND AYURVEDA.—Attempts have been made in the past to suggest integration of both systems of medicine *i.e.*, modern system—so-called Allopathic system of scientific medicine and Ayurveda, our indigenous and ancient system of medicine, by appointing Chopra Committee by the Central Government and Vodh Committee by the Bombay State. In September, 1950, a conference of the Health Ministers of all States was held in New Delhi and their recommendations of the teaching of Physiology, Anatomy, Radiology and other scientific subjects in Ayurvedic schools both for diploma and degree courses were opposed very strongly by the Governing Council of All-India Ayurvedic Congress, who passed the following resolution at their meeting held recently in New Delhi:—

> "It is the firm conviction of the All-India Ayurvedic Congress that the linking of Ayurveda with preliminary studies of Allopathy will not work. It will not only confuse the brain of the students, but will also make the Ayurvedic education as costly and removed from the reach of the student of ordinary means as allopathy is. Moreover, the health problem of the country will remain unsolved to any satisfaction for long time to come."

If we analyse the mind behind the resolution it will be seen that the Governing Council seek a royal and easy road

to learn and practise Ayurveda in utter disregard of what may happen to the people for whose health they are clamouring so much. It seems that Vaidyas want to teach their students unalloyed and stale Ayurvedic science. They do not seem to realize the limitations of Ayurveda and do not wish to be benefited by the progress modern medical science has made during recent years. How are the Vaidyas depending on Ayurvedic science going to deliver a transverse presentation of a fœtus, or relieve an acute intestinal obstruction due to a constricting band? I am reminded of a case of retention of urine in a young lady, the daughter in-law of the then famous Vaidyaraj, who was suffering from severe burns. I was consulted for pain over the lower part of the abdomen. Pain was due to acute retention of urine which was not diagnosed by the Vaidyaraj. No sooner did I offer the diagnosis of retention of urine, than he jumped up saying that a dose of his medicine will relieve the bladder immediately. I humbly inquired of him if there was any thing wrong in catheterizing the patient so that she might be relieved at once. It was not so much the remedy as much as the diagnosis. One must first know what he is going to treat. I am afraid they show gross ignorance about the human sufferings when they even resent the basic knowledge of Anatomy and Physiology—the knowledge that tells you only the structure and functions of the human body. Is it possible even to treat an abnormal condition, called disease, when one does not know the normal functions of a body? There cannot be two anatomical structures, *viz.*, ancient and modern, nor can there be similarly two physio logical systems. There may be certain things still in the normal functions of the human body which may not be understood by us, but that should not debar us from acquiring the knowledge of facts known to us. I am afraid they do not show progressive mind when they want to stick to the old static science wherein no progress and research have been made for over a thousand years. However, I agree with them that when it comes to clinical teaching, the student's mind is likely to be confused when two systems are presented together, unless he has mastered at least one system. I therefore submit that the establishment of separate Ayurvedic schools will not help us deliver the goods. Medical graduates will be the proper persons to synthesize both these systems by *post-graduate studies in Ayurvedic medicine.*

When they suggest that separate State Directorates of Ayurveda manned exclusively by Ayurvedic pandits be appointed, I am afraid they are asking too much, especially when they have no idea of preventive medicine. It is quite evident that they are thinking more of power than the welfare and health of the community in whose name they claim the favour of the government.

They also talk about the high cost of medical education and treatment, but let me remind them of what our Prime Minister said at the recent All-India Surgeon's Conference held in New Delhi in this connection, "Death is the cheapest of them all." I humbly ask why then try any remedy at all?

Under these conflicting conditions it has been my pleasant duty with my other colleagues, namely Dr. Bhasker Patel of J. J. Hospital, Bombay, Principal G. V. Barve of B. J. Medical College, Ahmedabad, and Vaidyaraj Vallabhram of Ahmedabad to inspect and report on the proposed course of M.B.S.S. and Ayurveda at Jamnagar Ayurvedic Medical College on behalf of the Gujarat University. Fortunately, the spirit behind the move at Jamnagar is an enlightened soul trying his best to get the good out of the old, with an open mind to the modern medical science in which he is fully trained. He suggests that Anatomy and Physiology should be taught during the first two years. There can be no two opinions about it, though as I mentioned above, All-India Ayurvedic Congress opposes it. The question of teaching Ayurvedic materia medica and medicine at which period of the course of study has yet to be decided. I think honest attempts must be made to arrange this curriculum in such a way that one system does not suffer. One year more is suggested for Ayurvedic course and there is a feeling that it will be amalgamated.

Vested interests apart, the curriculum may be given a fair trial to satisfy public opinion and let truth be brought out from the hoary past. Nothing will be lost, but some good may come out of it.

If we succeed, Jamnagar will be the first to amalgamate both the systems and Gujarat University will be the first to help such amalgamation.

REGISTRATION OF MEDICAL PRACTITIONERS.—The ways of democracy are unscrutable. In 1938 all the unqualified medical practitioners practising as Vaidyas and Hakims and even some compounders of the Bombay State were registered under the Bombay Medical Practitioners' Act, 1938, and the public were assured at that time that no more registrations would follow. New entrants would have to undergo medical training for a certain period and hence the unqualified practitioners will disappear. But once more unqualified practitioners who are supposed to practise as vaidyas, hakims, bone-setters, etc., in the towns and cities of merged States and similar displaced practitioners from Sind, West Punjab and East Bengal who have been here as refugees are to be registered as medical practitioners under the Bombay Medical Practitioners' Act of 1938.

The advantage of this, I need hardly say, is being taken by ward boys, assistants of compounders, laboratory boys and such other fortunately placed persons including Ayas and unqualified nurses, to be dubbed as doctors and to sit and practise with those who have worked and studied the full medical curriculum of the present medical colleges.

In this country, it is very easy to get a certificate from a so-called respectable person or even an officer regarding the period of medical practice by a particular person. I would therefore suggest to the authorities to scrutinize thoroughly the statements made in their application forms before they are registered as medical practitioners. I shudder to imagine the fate of the accused persons charged with criminal offences under the Indian Penal Code, in which expert medical evidence is required for the administration of justice, if the judges were to rely upon the expert testimony of such registered medical practitioners, as under section 33 of the Bombay Medical Practitioners' Act they are considered as qualified to give evidence at any inquest or in any court of law as experts under section 45 of the Indian Evidence Act, 1872, on any matter relating to medicine, surgery or midwifery. You will agree with me that such a procedure will lead to a travesty of justice.

PRESS ADVERTISEMENTS.—Advertisements not only by quacks but even by some medical practitioners registered under the Bombay Medical Practitioners' Act 1938 of all sorts of quack remedies ranging from promise of pregnancy

to a barren woman to a cure of impotence are being published in the lay press. Such advertisements are contrary to the public interest and discreditable to the medical profession, and any registered medical practitioner resorting to such unethical practice renders himself liable to have his name removed from the Medical Register.

SOCIAL WORKER AND THE HOSPITAL.—Our public hospitals sometimes come into a good deal of unfair criticism either in public, press or in private. Government and public bodies spend a large amount of money to relieve the suffering public. Whatever good is done in such institutions is rarely appreciated. If a certain thing which is expected by a patient is not done according to his desire, he at once rushes to the press which publishes imaginary grievances and allegations without adequate substantiation. In my humble opinion such behaviour of the press is likely to do more harm to the public in whose interest they are supposed to act. Rumours are even more powerful than the press, as the credulous public are inclined to believe them, which are always spread widely with great exaggeration, while the physician or surgeon concerned is bound by certain rules of service and medical ethics and gets no opportunity to explain his point of view. The indiscriminating public suffers in the end.

All this, to my mind, can be avoided by engaging a social worker in a public hospital. He will see the patient and his relatives, discuss their problems and difficulties and will advise and guide them. As a non-medical person he will explain the nature of the disease with sympathy and human understanding and the result of treatment either medical or surgical and the after-treatment to be followed later on for the patient's benefit. The difficulties of the patient or of his relatives will be properly placed by such a social worker before the medical or the administrative staff of the hospital. Thus, a mutual goodwill will be created between the patient and the hospital staff. The patient then becomes the enthusiastic admirer of the institution, and ultimately its benefactor to the extent to which his existing or future means will permit. The hospital staff will thus also be treating not the disease but the diseased body and mind—the ultimate aim of all medical aid. I believe that the Tata School of Sociology trains such types of individuals, who may be engaged in big public hospitals.

PREVENTIVE MEDICINE.—We are all giving more attention to the cure and less to the prevention of disease. It is left to the health officer of a municipality if one exists, to do this part of the job. No doubt, he is a specialist in Public Health and he does it to his best ability, but we as medical men cannot shirk our responsibility.

We can help individually by reporting our infectious cases to the proper authorities. Besides, as members of the branches of the Indian Medical Association we should take up this question. Propaganda for the prevention of diseases by slides, charts and lectures and demonstrations in every town and even in every mohalla of a city where a member resides will go a long way to create goodwill between the physician and the people wiom he has the honour and privilege to treat.

I may mention that the Ahmedabad Medical Society is shortly going to make a sample survey for finding out the incidence of various diseases amongst textile workers, and individual examination of each and every worker in respect of those diseases in which immediate help can be given. Each member of the Society will spend two hours twice a week for a period of one year to complete this work. There are 130,000 labourers in Textile Industry and when this survey is completed, important data will be available. In this self-imposed task the Medical Society will have the help and co-operation of Ahmedabad Textile Industrial Research Association, Ahmedabad Mill Owners' Association and the Ahmedabad Textile Union. Such a survey may also be carried out in other industrial cities of our State.

One more important and yet not much published activity is done by the trustees of Kasturba Gandhi National Memorial Trust whose work is mainly confined to women and children. There is a training centre at Koba near Ahmedabad, where young ladies are trained for a prerequisite course for working in the rural areas. They afterwards get their midwifery training at Vadilal Sarabhai General Hospital and Chinai Maternity Home, Ahmedabad. They are posted in the villages where the population is less than two thousand. Here they act as friends, philosophers and guides to the village people not only in medical matters but also in their other activities.

TUBERCULOSIS IN GUJARAT AND SAURASHTRA.—Though no actual figures are available, it is the general opinion of the medical practitioners in these areas that pulmonary and other types of tuberculosis are very common. Some assert that 40 per cent of their practice consists of these types of cases.

The institutional treatment for these types of cases is now well established. Unfortunately, such institutions are only in Baroda and in Saurashtra. There is some work done at Anand. This is nothing as compared with the prevalence of the disease. Even in these institutions more paying patients are admitted than the poor.

It is earnestly suggested that the Government should start at least a big institution for the treatment of these cases in this part of the country. I may also suggest here that tuberculosis is fairly common and rapidly spreading among workers in Textile Mills. The Labour Union should also direct its attention to this problem.

HONORARY PHYSICIANS AND SURGEONS.—They are now engaged by several institutions in cities in Bombay State. The Saurashtra Government are requested to adopt this system as their cost of administration will diminish and the public will have the benefit of the advice of experts.

* * * * *

The following resolutions were passed in the 5th Gujarat and Saurashtra Provincial Medical Conference held at Nadiad:—

1. This Conference places on record its deep sense of sorrow at the sad demise of three great sons of India *viz.*, Shri Aurobindo Ghosh, Sardar Patel and Shri A. V. Thakkar (Thakkar-Bapa), who have contributed all they can towards the making of the Nation spiritually, politically and socially at the cost of their lives.

This Conference places on record its deep sense of grief and sorrow at the sad and untimely demise of the following members of the Provincial Branch:

1. Dr. K. S. Apte, Baroda.
2. Dr. G. O. Shah, Ahmedabad.

2. In view of the fact that social medicine plays an important role in the prevention of illness, cure of disease

and maintenance of positive health and that an understanding of the socio-economic problems and environments of a patient is essential for sound diagnosis and all the technical procedure available to the physician and surgeon, it is hereby resolved that all local Bodies, Governments of Bombay State and Saurashtra be requested to take early steps to appoint trained social workers in big public hospitals whose duty will be to render every kind of assistance to ameliorate the sufferings of patients admitted to these hospitals by contacting them and studying the social and environmental conditions under which they live and work.

3. Whereas some medical practitioners registered under the Bombay Medical Practitioners' Act 1938 are found to advertise in the lay press for their personal gain, it is hereby resolved that the Governments of Bombay State and Saurashtra be requested to direct the Board of Indian Systems of Medicine formed under this Act to exercise its disciplinary function over medical practitioners registered under the said Act, to publish a code containing the main principles of medical ethics for their information and guidance and to issue warning notices comprising certain unethical practices which are regarded as falling within the meaning of the term, infamous conduct in a professional respect.

4. This Conference requests the Universities of Bombay, Gujarat and Baroda to remove Intermediate Science Examination bar for such medical licentiates of at least ten years standing as are desirous of undergoing the condensed course of study for the M.B.B.S. degree examination.

5. Resolved that this Conference is strongly of opinion that medical science should be taught in Hindi. It further requests the Universities in Gujarat to replace English by Hindi as medium of instruction within a period of not more than fifteen years and further to use international terminology using Hindi wherever feasible.

6. This Conference notes with pleasure that the World Health Organisation (W.H.O.) has allotted to the Bombay State a sum of Rs. three lacs for research in tuberculosis in industrial areas.

This Conference requests the Government of Bombay to utilise this amount for the said purpose in highly industrialised areas.

MADHYA BHARAT MEDICAL CONFERENCE

The Madhya Bharat Provincial Medical Conference was held at Mhow on Sunday the 1st July 1951. Due to the sudden death of the Honourable Sri R. S. Sharma, Minister of P.W.D. and ex-Minister of Health, the Conference had to be postponed from the 30th June to the 1st July and the Hon'ble Chief Minister regretted his inability to be present at the Conference.

Hon'ble Sri Ram Sahay, Speaker of the Madhya Bharat Legislative Assembly inaugurated the Conference. In his address, he stressed the immediate need for manufacturing all drugs in India and for research in Ayurvedic medicines.

Dr. Premsingh Rathor, the Honourable Health Minister inaugurated the scientific session of the conference. In his speech, the Hon'ble Minister stressed the importance of the scientific outlook and scientific research. He said " the various scientific researches that have brought the medical science to the present stage have only given a hint to the scientists regarding the innumerable hidden things that are still to be brought to light." He informed the house that the Government of Madhya Bharat were keen to arrange for necessary facilities in the two medical colleges of the State for building up research units.

The medical department had recently taken up a scheme for refresher courses. A scheme for starting a drugs laboratory was also engaging the attention of the Government. Dr. Rathor added that the financial resources of the Government for medical and health activities and for medical research being limited, generous donations from the public and the active co-operation of the medical profession were essential for an all-round improvement in health matters.

DR. S. K. MUKERJI,
President, Madhya Bharat Provincial Medical Conference.

The following are excerpts from the presidential address delivered by Dr. Mukerji:—

I am fully conscious of the heavy responsibility you have placed on my slender shoulders by electing me as the President of the Indian Medical Association, Madhya Bharat Province. We are passing through difficult times and have numerous problems before us which need satisfactory solution. We can no longer blame the foreign Government for their past omissions and commissions. As citizens of a free country, we have to take the entire responsibility on ourselves. We can make or mar our own future. Our association has an important part to play in shaping the policy of medical relief and public health in our State by suggesting practicable proposals and offering constructive criticism to our Government. We have to mobilise public opinion, create health consciousness amongst our own people and secure funds from the philanthropic associations and the charitably minded persons to aid the implementation of our health schemes.

MEDICAL RELIEF

The problem of medical relief deserves our close consideration. Though considerable efforts have been made by

the Government of our newly formed State to improve the facilities for medical relief, yet the conditions leave much scope for improvement. We are aware that all the district hospitals have now been equipped with x-ray plants and some instruments and laboratory equipment have also been provided. But the provision for equipment is not enough. Suitable trained personnel must be provided who could usefully utilise the equipment for public benefit. Our aim should be to organise in such a manner that each district hospital can work as a self-sufficient unit capable of efficiently handling all the work of the district. Each civil surgeon should be a reasonably good surgeon. Where a trained surgeon is not available the prospective civil surgeon should be given facilities for training for a minimum period of three months in one of the two hospitals attached to the medical colleges. Each district hospital should also have a properly trained physician, a clinical pathologist, a radiologist and a lady doctor. Suitable training facilities for the above personnel should be available in the hospitals attached to the medical colleges. With the above objects in view, a scheme should be drawn up on the basis of a five-year plan, and the implementation of the scheme should be started as soon as possible. The services of suitably qualified medical men as honorary medical officers, whenever available, should be utilised in the district hospitals. While the urban medical relief needs improvement it is the village medical relief on which every well-wisher of our State and the Government should concentrate. It is heartening to note that our Government have already moved in the matter. We would earnestly request the Government to adopt as soon as possible a scheme of subsidised medical practice. We suggest that to begin with, serious attempt should be made to replace the compounders in charge of dispensaries with qualified medical men on a subsidised basis. These days the financial position of the farmers in the villages is better than it used to be before the world war no. II and it seems that it is not unlikely that given suitable terms, it would not be impossible to attract young qualified men to settle down in the villages where they would be able to earn comfortable living. The same person would be able to look after the sanitation of the village and implement the health schemes sponsored by the Government. An educated well-meaning doctor is likely to command much more respect than any other person of the village and may perhaps achieve the seemingly impossible. Perhaps the financial implications of this scheme are not beyond our means. At least a start could be made without any undue delay in certain areas, where the plan is likely to work. The scheme may subsequently be extended to other areas.

Public Health

The public health is equally important if not more so. In my daily practice I have often been amazed at the colossal ignorance of the elementary principles of health even amongst our so-called intelligentsia. Every one of us, without any great effort or much loss of time, can help to mitigate this deplorable state of affairs and during our routine visits to our patients, mobilise health consciousness among our people by instructing them about personal environmental hygiene and prevention of the disease from which the patient is suffering. We should also request the Government and the different boards of studies to introduce Hygiene in the curriculum of studies in the primary and secondary schools as a compulsory subject. Arrangements should be made by the health department to show cinema films, occasionally, in the primary and the secondary schools and the colleges. illustrating the preventive aspects of the common diseases of our State. Similarly in urban areas where there is a public cinema, arrangements may be easily made with the cinema proprietor to occasionally project, during their routine shows, short films illustrating the preventive aspects of the diseases that are common in that place, during that time of the year. In the villages, the doctor, the schoolmaster, and the educated young men can do a lot in developing the health consciousness among our people. The mobile units of our health department can show films and posters which would be interesting and at the same time instructive to the villagers.

Medical Education

We have now two medical colleges in Madhya Bharat. We sincerely thank the Government for taking over the Mahatma Gandhi Memorial Medical College and the associated institutions. Admission in these colleges should strictly be on merits so that we get the right type of material for training as doctors. Throughout the training period the teachers should concentrate on the practical aspects of their subjects and try to develop in the students their independent line of thinking, initiative, and correct approach to the problems they will have to face in their future career. Our examinations, however, should be so conducted that a candidate cannot pass his examination by simply pursuing the cramming up technique without intelligently understanding his subject.

Both the medical colleges should have ample facilities for postgraduate training and research. We suggest the formation of special units in the medical colleges. For instance, we may have a chest unit, a cardiovascular unit, a nutritional unit and a hæmatological unit at Indore. At Gwalior we may have a neurological and neurosurgical unit, a psychological unit, a gastrointestinal unit and a kidney and a urogenital unit. Some trained personnel are already available in these places. Suitable young men, may be trained up, who could later on be sent up for advanced training abroad at State expense.

As a person who has been connected with teaching for the best part of his life, I believe that in the matter of selecting our teachers for any educational institution, the best available talent should be employed without any consideration of caste, colour, creed or provincialism. This policy should particularly apply to the matter of selection of the senior staff of any teaching institution. Bright young local men could then be trained up under the guidance of these men to be the future professors. Indeed, if we had the means, I believe that it is a good investment to employ if available, men of international reputation as our professors on high salaries for at least a period of five years. It will fetch rich dividends later on.

Training of Nurses and Midwives

No hospital can run efficiently without an adequate and efficient nursing staff. This problem, as you can well imagine, is going to be our headache for some time to come. We have to offer better salaries, better housing, better food and arrangement for proper recreation to the nursing staff, so that we can attract suitable girls from the middle class families. Nurses' training should be at least of the B.P.N.A.

standard and, if possible, only matriculates should be admitted for the course.

Training of midwives should be speeded up. To begin with, candidates who have passed Class VII standard may be admitted to this course. Three months of training in medical and surgical wards and intensive training for one year in maternity and gynæcology wards would perhaps be the minimum period of training, that would be necessary for the midwives. Training of these midwives may be undertaken and encouraged in the Government district hospitals where suitable facilities exist, as well as in suitable non-Government hospitals, *e.g.*, the Mission hospitals and maternity homes. Better salaries and prospects should be offered to these midwives. After they pass out they should be absorbed in as large number as possible in Government service and be sent out to the outlying dispensaries of our State where they could do antenatal and obstetric work. This would be the quickest, cheapest and the best way for the time being, which would enable us to reduce the maternal and infant mortality in the rural areas.

I would like to draw your attention to another subject which has so far not received adequate attention from the authorities. This is the question of training up of technicians. Lot of the routine work in the laboratories and x-ray departments can be partly done by well-trained technicians, leaving the medical officer more time to attend to other work which needs more of his specialized skill. I suggest that in the future set-up, the technicians should at least be matriculates and be appointed on a higher scale of pay than the compounders.

One Word about the Association

At present there are 15 branches affiliated to the Indian Medical Association through our Madhya Bharat Provincial Branch, which includes branches at Bhopal and at Rewa (Vindhya Pradesh). I wish there should be at least one branch in every district, and all the doctors of Madhya Bharat should feel it obligatory to join the Indian Medical Association through their local branches and strengthen the hands of the parent body so that it could help them with all its force.

I suggest that every branch must have at least one clinical meeting every month in which important cases must be discussed and occasionally papers on important subjects should be read. From time to time lectures by the professors of the medical colleges of Madhya Bharat should be arranged. This will help every practitioner in service and practice to brush up his knowledge. The membership of the Indian Medical Association in Madhya Bharat is now 400, I hope that it will rise much more in the near future.

Unauthorised Medical Practitioners

It is high time that some legislation be enacted to prevent the unqualified men from practising quackery amongst the credulous public. A provincial register of all the medical practitioners, qualified to practice should be made. We know that there would be quite a lot of difficulty in dealing with vaids, hakims, homœopaths who may not have suitable qualification. For enlistment, a certain amount of reasonable leniency may have to be observed in the beginning but only the fully qualified should be admitted in the register subsequently.

I may be pardoned for venturing to express my opinion on the difficult problem of the "other systems of medicine." I have only to submit that a false sense of patriotism or sentiment or expediency should not be allowed to cloud our vision. We should have a rational and logical approach to a vital problem of this nature. I am afraid that the policy of trying to integrate the eastern and western systems of medicine with their widely divergent basic principles, will not work. It will confuse the student. He will find it exceedingly difficult to correlate the theory of 'Vayu,' 'Pitta,' and 'Kapha' with the modern conception of the pathology of diseases.

Good health of the people is the best asset of a nation and as such it is very necessary for any Government to provide liberally for the department of medical relief and public health in its budget. We sincerely hope that the Government would be able to set aside a larger part of its revenue for medical relief and public health in their future budgets.

We are very grateful to our Hon'ble the Chief Minister, and Hon'ble the Health Minister, who in spite of their numerous engagements, found time to be with us this evening. We deeply appreciate their efforts for the improvement of medical relief and public health of our State and we assure them of our fullest co-operation in their arduous task.

* * * * *

The following resolutions were passed at the Madhya Bharat Provincial Medical Conference at Mhow in July 1951:—

1. (*a*) This conference of the Madhya Bharat Provincial Branch of the I.M.A. places on record its deep sense of sorrow at the sad demise of (*i*) Hon'ble Shri R. S. Sharma, Minister or P.W.D., and formerly Minister for Health, (*ii*) Sardar Vallabhbhai Patel and (*iii*) Dr. K. S. Ray, past President of the I.M.A. and the Editor of the J.I.M.A.

(*b*) This Conference also mourns the sudden and shocking death of Dr. K. K. Verma of the Madhya Bharat Medical Service who met with a motor accident while proceeding on Government duty.

2. This Conference urges the Madhya Bharat State Government to exempt the registered medical practitioners from the operation of the Sales Tax as is being done in Bombay, West Bengal, Madras and East Punjab.

3. This Conference requests the Madhya Bharat Government that the grade of the medical licentiates be revised from Rs. 150 to Rs. 300 without having any efficiency bar of selection posts. They are further requested that at least 10 per cent posts of the assistant surgeons be reserved for the worthy medical licentiates.

4. (*a*) This Conference requests the Medical Council of India that facilities for the admission of the matriculate licentiates to the Condensed M.B.B.S. Course be extended up to 1958. It further resolves that the universities be requested to reduce the training period of the Condensed M.B.B.S. Course from two years to eighteen months.

(*b*) This Conference thanks the Madhya Bharat Government for starting the M.B.B.S. Condensed Course at Indore and requests them to see that the number of admissions for this course be increased as much as possible to enable the licentiates to obtain a medical degree.

5. This Conference reiterates the following resolutions passed at the last provincial conference held in 1950, and

recommends to the Government to implement these at an early date:—

(*a*) This Conference requests the Madhya Bharat Government to introduce a refresher's course at the M.G.M. Medical College, Indore, as well as at the G. R. Medical College, Gwalior, from this year, if possible, so that a large number of members of the medical profession and also those in the Madhya Bharat Medical Service, for whom it should be made compulsory every few years, may get benefit from this course.

(*b*) The Conference notes with regret that certain essential drugs are being misused to a very great extent and poisonous drugs continue to be used by unqualified persons and sold without restrictions. These drugs should be available only on prescriptions of the registered medical practitioners of the modern scientific system.

(*c*) This Conference also urges on the Madhya Bharat Government the necessity of implementation of the provisions of the Drugs Act, 1940, and the Pharmacy Act, 1948, and of enactment of the Medical Practitioners' Registration Act in Madhya Bharat and the formation of a Provincial Medical Council.

(*d*) This Conference is alarmed to notice that the smoking habit amongst the juveniles is on the increase, and as such it recommends to the Government of Madhya Bharat that they should take such measures and enact laws as they deem fit to prevent this menace to juvenile health.

* * * * * *

AMBALA BRANCH—The annual functions of the branch was held on the 28th September 1951 at Dr. Gibbon's residence and on 29th September, 1951 at the Civil Hospital. Several interesting papers were read and discussed and some films were also shown. Dr. I. Gibbons and Dr. O. P. Varma were elected as the President and the Secretary respectively.

ASSAM PROVINCIAL BRANCH—The Working Committee of the branch met on the 6th September, 1951 at the residence of Dr. H. K. Das in Gauhati. 11 members were present and Dr. H. C. Baruah presided. The Secretary of the Assam Medical Relief Committee stated that a donation of Rs. 10,000 was received from the Governor's Fund and 5 medical units were working for medical relief work. Dr. B. Barua was declared as the duly elected President of the branch for the year 1951-52.

AZAMGARH BRANCH—At a special meeting of the branch held on the 9th September 1951, the members condoled the sad death of Dr. I. H. Rizvi.

CHAPRA BRANCH—A meeting of the branch was held on the 22nd April 1951 at the residence of Dr. G. B. Sahay. Dr. Sahay spoke on medico-legal subjects.

The annual general meeting of the branch was held on the 29th September 1951 with Dr. A. B. P. Sinha in the chair. The Secretary stated that the monthly meetings have been held regularly. Dr. A. B. P. Sinha and Dr. L. P. Verma were elected as the President and the Secretary respectively.

COIMBATORE BRANCH—A meeting of the branch was held on the 25th August 1951 with Dr. M. G. Nair in the chair. 78 members were present. Dr. D. Prosad spoke on "the G.P. and his T.B. patient." Some interesting films were then shown.

GHUGUDANGA BRANCH—A meeting of the branch was held on the 26th August 1951 with Dr. S. Sarkar in the chair. 19 members were present. There was an interesting lecture and discussion on "Abortion and its management by general practitioners."

FAIZABAD BRANCH—The annual general meeting of the branch was held on the 30th September 1951 at the municipal hall with Dr. (Kumari) V. V. Dube in the chair. 33 doctors were present. Dr. S. P. Gupta spoke on "The Common Eye Diseases in General Practice." Dr. H. N. Gupta and Dr. Y. D. Kapur were elected as the President and the Secretary respectively.

FEROZEPORE BRANCH—At a general meeting of the branch held on the 24th April 1951, it was decided to observe "Anti-fly week" in Ferozepore City and Cantonment from 23-5-51 to 29-5-51. The programme consisted of lectures in teaching institutions, processions of school children, cleanliness campaign, inspection of places dealing with eatables, and demonstrations of appropriate cinema shows and exhibitions.

Shri N. K. Mukharji, I.C.S., Deputy Commissioner of Ferozepur appreciated this very useful move of the medical association and gave his whole-hearted and active support to it. The branch at its meeting held on the 10th July 1951, with Dr. Rajaram Bhola as the President, also thanked other Government and Municipal officials and the public for their co-operation and assistance to make this public health activity of the Association successful.

JAGATDAL BRANCH—A meeting of the branch was held on the 14th August 1951 at Bhatpara Municipal Hall with Dr. S. Chakraburtty in the Chair. Dr. S. K. Roy spoke on "Marasmus in Children."

MADURA BRANCH—A meeting of the branch was held on the 4th August 1951. Dr. Miss Erickson spoke on "Some clinical aspects of Sterility."

* * * * *

At another meeting of the branch held on the 22nd September 1951 with Dr. M. N. Rajan in the chair. Dr. K. Balakrishnan spoke on "The Tuberculosis Problem in Madurai" Dr. J. Kvittingen referred to the work to be done under the auspices of the W.H.O. particularly regarding serological testing of groups of population.

MALEGAON BRANCH—A meeting of the branch was held on the 22nd July 1951 at the residence of Dr. M. K. Bodas. Dr. V. B. Davalbhakta spoke on "Common Ear Diseases and their treatment."

* * * * *

At the annual general meeting of the branch held on the 23rd September 1951 at the residence of Dr. G. Jahagirdar. Dr. V. R. Gadgil and Dr. G. S. Parnaik were elected as the President and the Secretary respectively.

MANGALDAI BRANCH—At an inaugural general meeting of the members of the medical profession of Mangaldai and its neighbourhood, with Dr. J. Chakrabarty in the chair. Dr. P. C. Duarah the Hony. Provincial Secretary of Assam impressed on the doctors present that in their interests at least it was essential to form a local branch of the I.M.A. at Mangaldai.

After the necessary formal resolutions were passed, Dr. B. Das and Dr. M. A. Ali were elected as the President and the Secretary of the newly formed branch.

NABADWIP BRANCH—At the annual general meeting of the branch held on the 5th September 1951, Dr. M. L.

Kundu and Dr. A. N. Maitra were elected as the President and the Secretary respectively.

PORBANDAR BRANCH—A meeting of the branch was held on the 8th July, 1951 with Dr. H. H. Chavda in the chair. 17 doctors were present. The following clinical subjects were discussed at the meetings:—(i) Clinical Manifestations of Exophthalmos, (ii) Digitatis in Heart Disease, (iii) Impetigo Contagiosa and (iv) Blood Transfusion.

* * * * *

At another meeting of the branch held on the 5th August 1951, 27 doctors attended. The following clinical subjects were discussed at this meeting: (i) Diarrhœa in children, (ii) Varieties of Common Cold, (iii) Cæsarian Section and (iv) Chorea. X-ray plates and specimens of some other cases were also demonstrated.

* * * * *

The next monthly meeting of the branch was held on the 9th September 1951 and 25 members were present. By a resolution passed unanimously, the branch raised its annual subscription from Rs. 12 to Rs. 15 per member per year. Dr. H. H. Chavda and Dr. B. D. Jhala were re-elected as the President and the Secretary of the branch for the year 1951-52. The following clinical subjects were then discussed:

(i) Hæmorrhage in Enteric Fever (ii) Diagnosis of Common Fevers and (iii) Phrenectomy in Tuberculosis.

SULTANPUR BRANCH—At a special general meeting of the branch held on the 13th August 1951 at the District Hospital, the members condoled the sad death of Dr. Sadiq Ali who was a popular Vice-President of the branch and who expired on the 6th August, 1951.

TANJORE BRANCH—A meeting of the branch was held on the 29th July 1951 with Dr. E. K. Menon in the chair. 41 members attended the meeting. Clinical discussions were held on "recent advances in therapeutics."

* * * * *

At another meeting of the branch held on the 25th August 1951, 25 members were present. Clinical discussions were held on "Infections of the Hand." Capt. G. S. K. Iyer was felicitated for his decision to move to Rishikes to serve the Swami Sivananda's Mission as a surgical specialist, in an honorary capacity.

TIRUNELVELI BRANCH—A meeting of the branch was held on the 25th August 1951 and 39 members were present. After the routine business, Dr. Miss F. Kantayya spoke on "A day in a Singapore Family Planning clinic."

UTTAR PRADESH STATE BRANCH—A meeting of the working committee of the branch council was held on the 12th August 1951 at Kanpur with Dr. A. C. Banerjea in the chair. Ten members were present. At the outset the members condoled the sad deaths of the following members:—(i) Dr. T. N. Gupta of Saharanpur, (ii) Dr. J. N. Srivastava of Gorakhpur, (iii) Dr. Sepal Mehta of Aligarh, (iv) Dr. T. S. Saksena of Shahjahanpur, (v) Lt.-Col. D. M. Sachdev of Allahabad, (vi) Major N. D. Banerjee of Kanpur and (vii) Dr. Amir Sahai of Moradabad.

The following resolution of the Lucknow branch was endorsed and the Secretary was requested to forward a copy of it to the Director of Health and Medical Services:—

"Resolved that a Discharge Certificate with short case notes be given at the time of discharge, to all in-patients of the hospitals giving clinical, pathological and radiological findings, diagnosis and any advice necessary for future."

The Secretary informed the Committee that the Government of U.P. had recently issued instructions regarding the annual medical examinations of 'rickshaw-pullers' and stipulated that such certificates should be issued only by medical officers not below the rank of 'assistant surgeons.' The Association objected to this sort of discrimination.

Dr. Mrs. B. Thungamma was declared elected as the President of the U.P. State Branch for the year 1951-52.

NEWS ITEM—Dr. Amrita Nath Roy, Assistant Director of the Indian Institute for Medical Research, Calcutta and an Assistant Editor, Journal of the Indian Medical Association, left on 31-10-51 for U.S.A. where he will join Duke University for specialising in hæmatology under Prof. Hansen **Pruss.**

XI BENGAL PROVINCIAL MEDICAL CONFERENCE

The 11th Bengal Provincial Medical Conference will be held at Satyen Roy Road, *Behala* on the *15th and 16th December* 1951. Dr. J. C. Banerjea will preside.

For further details, the organising secretary, Dr. B. Chatterjee of 477, Diamond Harbour Road, Calcutta—34 may be contacted.

XVI UTTAR PRADESH MEDICAL CONFERENCE

The 16th U.P. Medical Conference will be held at *Lakhimpur Kheri* on *25th, 26th and 27th December*, 1951. Dr. Mrs. B. Thungamma will preside.

For further details, the organising secretary, Dr. C. P. Saxena of Durga Nibas, Lakhimpur Kheri may be contacted.

XXVIII ALL-INDIA MEDICAL CONFERENCE

The 28th All-India Medical Conference will be held at *Ahmedabad* on the *17th, 18th and 19th February*, 1952. Dr. T. S. Tirumurti will preside. The annual meeting of the Central Council of the Indian Medical Association will be held on the 15th and 16th February, 1952 at Ahmedabad. All branch secretaries are requested to send the names and addresses of their delegates, members and visitors to the Conference Secretary by the 5th January 1952, so that the necessary *railway concession forms* for return journey on single fare, may be secured for the persons concerned before the 15th January, 1952.

For further details regarding the Conference, the organising secretary may be contacted at the Prarthana Samaj Hall, Raikhad, Ahmedabad.

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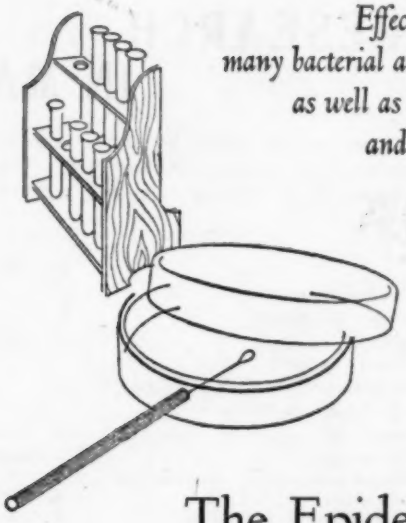

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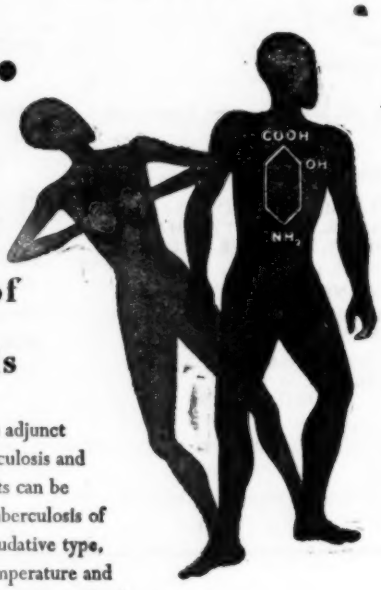

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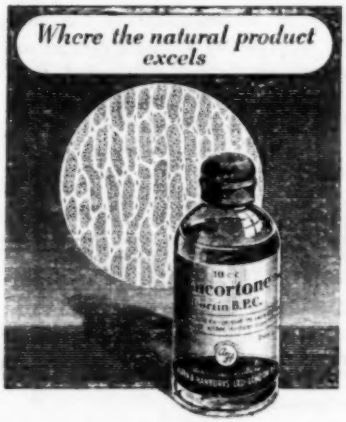

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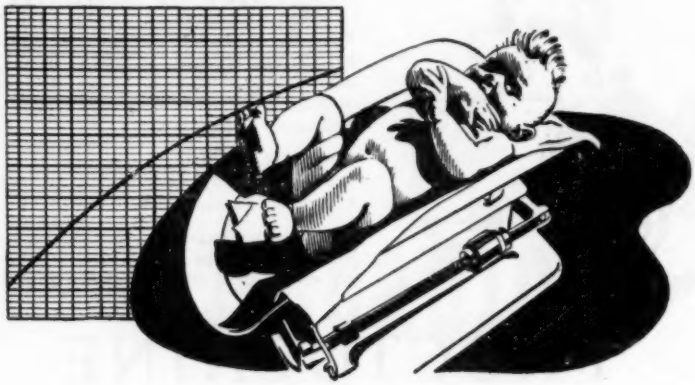

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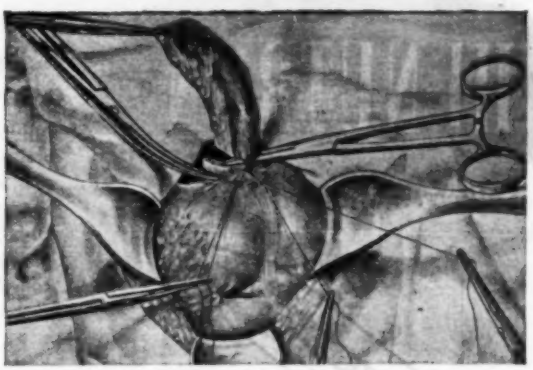

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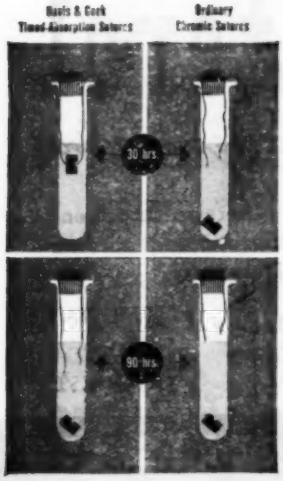

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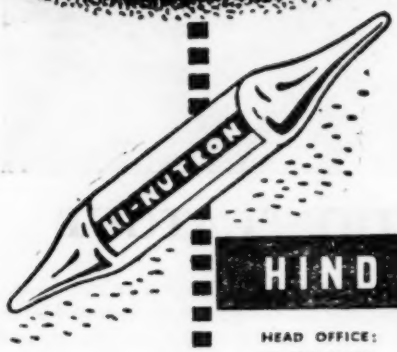

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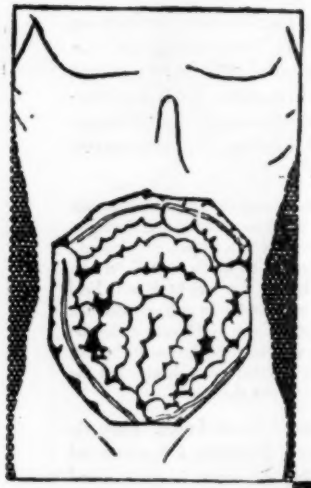

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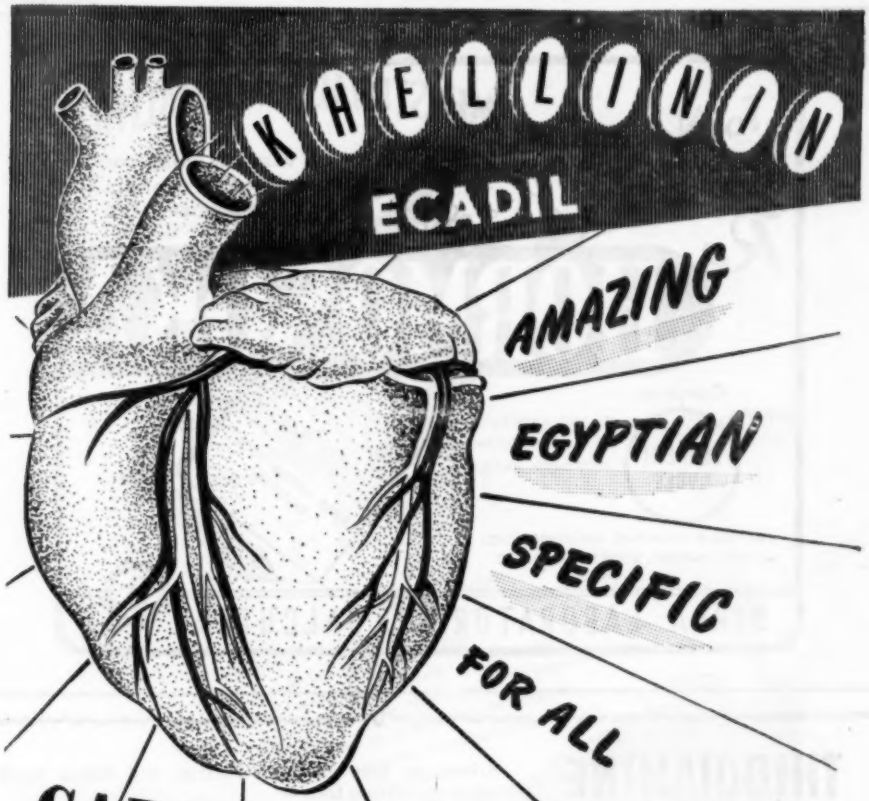

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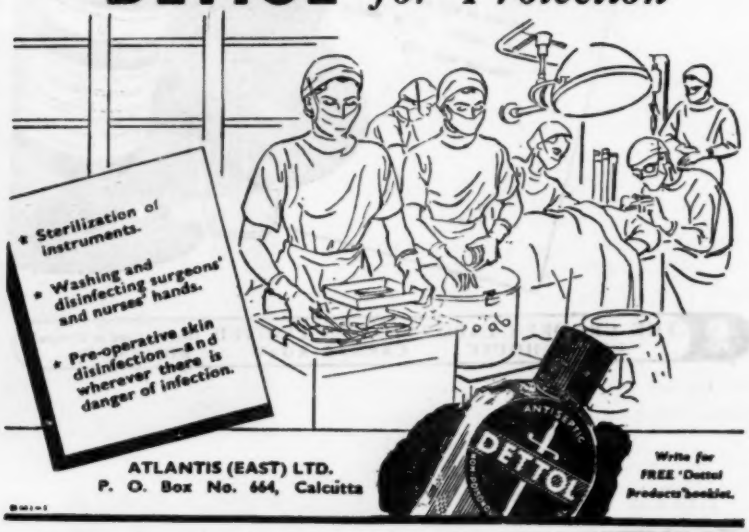
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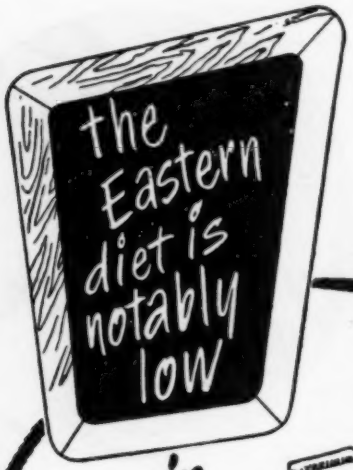

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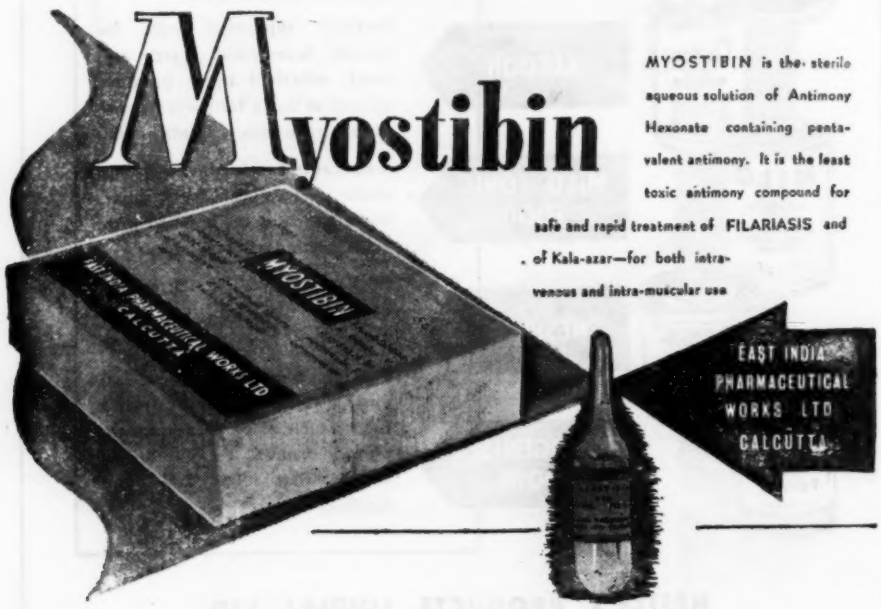

Journal of the Indian Medical Association

VOL. XXI. No. 3 CALCUTTA DECEMBER, 1951

ORIGINAL ARTICLES

CAUSE OF DEATH IN TRAUMATIC INJURY*

D. J. REDDY, M.D.,

Professor of Forensic Medicine, Andhra Medical College Visakhapatnam

"Those who dissected or inspected many bodies
Have at least learned to doubt,
When others, who are ignorant of anatomy
And do not take trouble to attend to it are in no doubt at all."

—*Morgagni.*

Injury due to trauma, accidental or homicidal in nature, is a daily event amongst civilian and military population and accounts for a large number of deaths. The traumatic agents may be blunt force; falls from automobiles, crushing or blast injuries; bullet wounds, burns etc. They may be minor and nonfatal or severe and mortal. Their immediate recognition and appropriate treatment are difficult problems facing the clinician. The pathologist finds it equally difficult when he has to account for the cause of death, with no morbid changes in the organs at autopsy.

We know that minor trauma like pleural puncture, sudden dilatation of anus, passing of a catheter, a blow over the abdomen or testis proved fatal long before medical aid could be summoned. Death in these cases is probably due to vagal inhibition which is reflex in nature (depressor nerve of the heart). Judges have been convinced on more than one occasion recently of this mode of sudden death due to minor trauma. Sudden deaths of similar nature had followed fright or sudden fall into water.

Yet in another group of cases, soon after trauma, the patient is in a state of peripherovascular collapse and from which he often survives, but the less fortunate few may succumb. This is due to neurogenic or primary shock, the painful nerve impulses often set up a reflex vasodilatation. But there is no alteration in blood volume. The pathologist, in these two types of deaths, result of trauma, can attribute to vagal inhibition or primary shock, only when a thorough postmortem examination reveals no pathological changes in tissues and as such is a diagnosis by exclusion. In concussion of the brain one may find no appreciable change in the central nervous system to account for death.

Larrey, Napoleon's army surgeon, in 1812 observed that soldiers sustaining crushing injuries with hardly any bleeding, developed after some hours a profound state of shock which we now recognise as delayed, secondary, traumatic or surgical shock. This is a syndrome initiated by diverse causal agents from trauma to toxæmias and burns, but presenting uniform and constant changes in organs and often proving fatal, if not attended to. Moon in 1942 has shown that there is a disparity between the volume of blood and the capacity of the vascular bed. Owing to liberation of a substance histamine-like in action but not chemically identical, at the site of trauma, permeability of capillaries is increased. This promotes effusion into tissues leading to reduction in blood volume aiding hæmoconcentration,—hæmatocrit and r.b.c. counts being abnormally raised.* At autopsy the organs are heavier owing to interstitial œdema, well seen in lungs and gastro-intestinal tract, cut surface unusually moist, congested, petechiæ over the mucous membranes, atony and dilatation of gastro-intestinal tract and parenchymal necrosis of the liver and the brain form the chief and characteristic findings.

Case 1—A woman aged 60, sustained compound fracture right arm which was amputated on 5-3-50 at K. G. Hospital. Her blood urea was 130 and 257 mgm. per cent on 7-3-50 and 20-3-50 respectively. Pulmonary œdema, sinusoidal dilatation and centrilobular necrosis of liver cells were the main postmortem findings in her, characteristic of secondary shock.

Crush syndrome, clinically indistinguishable from traumatic shock, in which renal failure accounts for death, although first reported amongst earthquake casualties in Messina in 1909 and 1914-1918 War by Germans, was revived and well authenticated with case reports by Bywaters and others in 1941 during

* Based on experiences of World War II and later in civilian life.

Read at the Vizagapatam Branch of I.M.A.

* Proved beyond doubt by Woodmansey at Burns centre; Comilla in 1945.

London blitz. In 1942-44 deaths due to uræmia of identical pathogenesis were reported amongst traffic and mining accidents, war injuries and casualties due to earthquake. The muscles at the site of trauma are crushed, and the patient passes into traumatic shock. Appropriate treatment leads to recovery. In some, anuria sets in, urine is dark-red due to myohæmoglobin and B.P. shoots up. A week later critical diuresis sets in with recovery.

In less unfortunate cases anuria continues, blood urea and potassium continue to rise and death occurs suddenly. In such cases, the traumatised muscles are swollen and bulge through their sheath when incised. The part is wet and œdematous. The muscle bundles are pallid, mottled with hæmorrhages, friable and necrotic in contrast to the surrounding normal healthy muscle. The kidneys are enlarged. They are pale and œdematous on section. The epithelium lining the ascending loop of Henle and second convoluted tubule is thinned out, ruptured and intraluminal hyaline casts are characteristically seen. Pigment casts of acid hæmatin, derived from myohæmoglobin are also seen in these tubules. (Benzidine test positive and identical with the pigment seen in urine). The kidneys prior to the onset of crush syndrome were normal and the changes described above are due to liberation of myohæmoglobin, or a toxin. The possibility that the renal lesions may be an allergic manifestation cannot be altogether ruled out.

In gun-shot wounds, death is often due to extensive bleeding caused by rupture of the main vessels. In Burma campaign of 1945-46, autopsies performed on gun-shot wound revealed large hæmorrhages due to rupture of carotid vessels or aorta or iliac vessels. Bullets have often been seen projecting through the rent in the vessel. Stab wounds and automobile accidents do lead to extensive bleeding. Profuse hæmorrhages due to endotheliolytic toxin of viperine bites lead to fatal end.

Case 2—Autopsy done on a man aged 60 (Jan. '50)—stab wound of the thorax, showed contusion and rupture of ascending aorta with hæmothorax.

It is difficult to say with certainty what is the exact amount of blood which must be lost to prove fatal although a loss of 1,500-2,000 c.c. in a short space of time may prove fatal. In air-raid casualties an acute loss of 3,500 c.c. was seen compatible with life for a few hours and even saved by timely and adequate blood transfusion. In deaths due to profuse bleeding the organs were pale, dry, shrunken and lighter with no petechiæ, a contrast to what one is used to see in deaths due to secondary shock at autopsy examination.

As against large extravasations of blood proving fatal, the neuro- and chest surgeons are familiar with the fatal compressive effects of small collections of blood in the brain, pleural and pericardial cavities. The embarrassment caused by hæmopericardium to the working of heart is too familiar to us.

Case 3—A male aged 35 was stabbed at 9 a.m. on 18-2-50 near Vizagapatam and expired on 19-2-50. Hæmothorax, collapse of the left lung with herniation of ruptured stomach into left pleural cavity were the main autopsy findings; illustrating the compressive effects of bleeding into pleural cavity.

Case 4—Female named G. aged 50 was hit with an axe over the head on 20-8-50 and came under the care of Dr. M. V. Ramanna Murthy, K. G. Hospital, Vishakhapatnam. Left pupil was dilated. Was in a moribund and comatose state. Expired on 22-8-50. Depressed fracture left parietal lobe, extradural hæmatoma of the size of a small orange on the left side (Fig. 1, *vide* Plate) and subdural hæmorrhage over the right cerebral hemisphere were the main autopsy findings.

Trauma round about the neck leads to asphyxial deaths due to blood clots blocking the upper respiratory passages and this mode of death I have seen in gun-shot wounds of the neck.

Yet another group of cases in which diffuse purpuric spots, in brain, or lungs, or liver, result of blast injuries, prove fatal. Sudden compression of chest leads to traumatic asphyxia, the upper part of body turning blue and mottled with purpuric spots.

Hæmorrhage caused by nonpenetrating trauma to internal organs often escapes our notice for characteristic signs and symptoms take time to manifest. Consistency of liver and its proximity to exterior leads to profuse bleeding even on minor injury, which may be subcapsular and masked or burst out and cause hæmoperitoneum. Splenic ruptures are more common than those of the liver and prove fatal in no time. In most of them, the spleen is found enlarged.

Case 5—A female aged 50 in Vizagapatam succumbed to fracture of the femur and the humerus. Surgeon suspected internal bleeding and autopsy on 22-12-49 revealed hæmoperitoneum caused by multiple tears of the spleen, some of them obliterated by blood clots.

In 1945, 46 and 47 autopsies carried out on soldiers stationed in Burma who sustained lethal injuries from motor cycle accidents showed extensive subdural and subarchnoid hæmorrhages with blood in lateral ventricles, yet showing an intact vault and base of the skull. Sudden deaths with a history of faint or fall are often reported as due to concussion. In the clinical state of concussion autopsy may reveal from no change to pulpification of the brain. But one frequently finds at autopsy that sudden death was due to rupture of an already atherosclerotic vessel or embolus having emanated from chronic valvular disease of the heart. In these cases the superficial scalp wounds often tempt the surgeon to ignore the hidden changes in the diseased organs.

Crushing injuries, or gross mutilation of organs, transection of the spinal column into two, basal fractures of the skull involving the midbrain and the medulla oblongata have caused instantaneous death,

all of which I had the misfortune to see in World War II. Death due to acute alcoholism is often made, ignoring the lethal injuries one had sustained under the influence of alcohol.

Case 6—Autopsy done on a Sikh sepoy in April '46 at Rangoon whose blood alcohol 12 hours after hospitalisation was 180 mgms. per cent revealed fracture ribs, surgical emphysema chest wall, rupture of spleen and hæmoperitoneum. The physician reported acute alcoholism as cause of death.

Aristotle recognised that a blow over the belly resulted in rupture of the bowel. Geoffrey Flarell (1949) has reported recently of how a cricketer went to bed and collapsed after sustaining non-penetrating injury to the abdomen. Multiple openings were seen in the duodenum on laparotomy.

Pockets of gas when spotted by radiography after non-penetrating injury to the abdomen is unequivocal evidence of rupture of the gastro-intestinal tract. Radiography is a valuable and reliable diagnostic aid to indicate rupture of the bowel in non-penetrating injuries to the abdomen. It often decides whether operative intervention is indicated or not.

Patients may recover from the immediate effects of trauma only to fall a prey to complications which although remarkably rare during World War II, needs to be considered. Thrombosis of the healthy or traumatised femoral veins, the formation of which is assisted by continued stay in bed, is well-known to the surgeons. The patient apparently progressing well suddenly spits blood, is restless and complains of pain in the chest, all due to pulmonary embolism having set in, due to dislodgement of thrombus from veins of the lower limbs. Embolectomy supplemented by dicumarol therapy could save the individual.

Case 7—Autopsy done in October 1945 at Rangoon in a gun-shot wound abdomen revealed mesenteric vein thrombosis as cause of death.

Although air embolism is an infrequent complication of trauma, it still is responsible for a small percentage of deaths in gun-shot wounds of the neck, artificial pneumothorax and air insufflation of the uterus or of the fallopian tubes and operations in the neck (subtotal thyroidectomy).

Case 8—A captain in the British army sustained injury of the head and died in July 1945 at Comilla immediately after blood transfusion. Autopsy done soon after death showed frothy blood in the chambers of the heart and air bubbles were seen in the left iliac veins, the site of entry of transfusion needle being in the vein adjoining the left medial malleolus. Intraluminal air bubbles in cerebral vessels or frothy blood in heart is often seen in bodies undergoing putrefaction and hence the absolute necessity of carrying autopsy examination soon after death, if air embolism has to be ruled out.

Long before fat embolism was realised as a cause of death in fractures of bones, Lower (1669), Magendi (1821) and Rudolph Virchow (1862) by introducing olive oil into animal circulation showed conclusively its destructive effects. In 1876, Holm quoted cases where fat embolism was not fatal and further by statistics proved that fat embolism is a common complication of fracture of bones and may often lack in clinical symptoms. It is realised now that fat embolism does occur as a complication of trauma of all adipose tissues (subcutaneous tissues) and the liver. In 200 autopsies carried on aviation accidents pulmonary fat embolism accounted in 31 per cent out of which 25 per cent showed demonstrable cerebral fat embolism.

Case 9—In December 1946 the urine of a sepoy with fracture of the right femur (52, I.G.H. Rangoon) showed fat globules and the patient had purpuric spots over the shoulder region which is one of the clinical manifestations of the condition.

At autopsy these cases show pulmonary hæmorrhages, œdema and the interalveolar vessels plugged with fat. Scraping (at autopsy) or puncture (biopsy) of lung tissue shows fat-laden capillaries after treatment with 2 per cent KOH. White matter of the brain often shows similar lesions. Frozen sections of these organs when suitably stained reveal the pathognomonic sudanophilic granules.

Intact skin and mucous membranes are impregnable to bacterial invasion. Trauma gives a beach head for invasive bacteria for their sinister operations. Streptococcal cellulitis, erysipelas, septicæmia and pyæmia are dreadful complications of trauma all of which can be controlled by sulpha group of drugs and antibiotics. Retardation of circulation and prolonged confinement to bed lead to broncho- or lobar pneumonia.

Pulmonary complications set in with great rapidity in victims of injuries to the head. Inadequate expansion of the lungs consequent on cerebral injury and the recumbent posture assumed by these patients promotes the development of pulmonary œdema and bronchopneumonia. In Case 10 and 11 bronchopneumonia was well-established within 8 to 12 hours after the head injury.

Case 10—A male named V.K. aged 45 was hit over the head on 7-10-50. He was unconscious and admitted under Dr. K. G. Krishnaswami, K. G. Hospital, Visakhapatnam. Expired on 8-10-50. Fracture right temporal and occipital bones; extradural, subdural and cortical hæmorrhages and well-developed bronchopneumonia of the lungs were findings at autopsy.

Case 11—S.R. aged 45 fell down from a railway engine on 16-4-51 and was unconscious. After 2 days' stay at K. G. Hospital, Visakhapatnam, under the care of Dr. Atma Rama Rao, he died on 18-4-51. Multiple abrasions over the body, contusion scalp over the parietal region, comminuted fracture vault of the skull, subdural hæmorrhage on the left side of the brain, and bronchopneumonia of both lungs were the autopsy findings (Fig. 2 *vide* Plate).

Secondary hæmorrhage, result of septic arteritis at or above the site of trauma, is difficult to control.

Case 12—A Chinese male aged 30 was admitted for compound fracture of the right arm caused by a gun-shot wound on 6-8-46 at Meiktila. The limb was put in plaster of paris and on 20-8-46 serosanguinous discharge was noticed. The wound was frankly septic by 26-8-46, bled profusely and the patient expired. Autopsy revealed foul-smelling, swollen and scaldy tissues of the right arm. The brachial artery buried in was friable and showed a rent with irregular edges.

* Penetrating head injuries—specially in the region of sinuses—due to gun-shot wounds have often lead to B. coli meningitis, encephalitis, ventriculitis or abscess, either alone or one passing into the other. The offending organism in all these cases was typical B. coli as typed by biochemical reactions, morphological and cultural appearances. (Figs. 4-8, *vide* Plate). One of them died of suppurative B. coli pericarditis while being treated for penetrating injury of the head, pericarditis not recognised antemortem.

Case 13—Child M. R. aged 8/12 was accidentally hit by by a stick while reposing in his mother's arms on 22-8-50. The child was under the care of Dr. K. G. Krishnaswami in K. G. Hospital, Visakhapatnam. Had convulsions over the right half of the body. X-ray skull revealed fracture of the occipital bone. Was running 101°F. temperature. C.S.F. was turbid. The child died on 24-8-50. Hæmatoma over left parietal region of the scalp, fracture occipital bone, suppurative meningitis over the superolateral surface and base of the brain and bronchopneumonia were the autopsy findings. (Fig. 3, *vide* Plate).

Although prophylactic inoculations have cut down the incidence of tetanus following trauma in the armed forces, tetanus of the parturient mother and the several wounded amongst civil life are not rare in the rural parts of our country.

Case 14—N.R.A., an adult male, sustained a fall on 16-5-50 and was treated for compound fracture of left arm at Anakapalle. He showed signs and symptoms of tetanus on 21-5-51 and was removed to K. G. Hospital, Visakhapatnam. Intramuscular paraldehyde and large doses of antitetanic serum were of no use. He expired on 22-5-50.

The deaths due to gas gangrene were remarkably low in World War II. This was in contrast to 10 per cent incidence of all wounds in 1914-1918 War. Pasteur, Robert Koch, and his American disciple Henry Welch were pioneers in the discovery of clostridial organisms responsible for gas gangrene.

Case 15—A sepoy while on patrol in Chindwin area in December 1942 was shot through his left leg. Two days later he was received at a field ambulance in Tamu. Compound fracture of tibia with marked swelling and emphysematous crackling was noticed. He was toxic. After amputation above the knee joint and antigasgangrene serum therapy, he was evacuated to casualty clearing station where he expired.

Peritonitis following non-penetrating trauma to abdomen are often being reported.

I have only presented to you a bird's eye view of the very vast subject of 'cause of death in traumatic injury' like what an air reconnaissance could offer useful but gross outlay of enemy territory, lacking in details. My purpose is more than served if we have reminded ourselves of how important it is to elicit careful case history and make accurate clinical observations for diagnosis and appropriate treatment of traumatic injuries. Functional states like concussion, or vagal inhibition or primary shock as cause of death in trauma are only tenable when a thorough and careful post-mortem examination showed normal organs.

* Observations made while pathologist to 3. British Neurosurgical Unit.

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MODERN INVESTIGATIONS AND MANAGEMENT OF INTRACRANIAL TUMOUR

R. S. SEAGAT,
Amritsar

The intracranial expansive processes consist of tumours and hæmatomas. The tumours group themselves according to certain familial resemblances of structure and behaviour. These can be classified as follows:

1. *Encephalic tumours.*

   a. *Gliomas*:

   1. Glioblastoma *multiforme;* 2. spongioblastoma *unipolare;* 3. ependymoma; 4. neuro-epithelioma; 5. astroblastoma; 6. astrocytoma; and 7. oligodendroglioma.

   b. *Glioneuromas*:

   1. Medulloblastoma and 2. ganglioneuroma.

2. *Tumours of the covering cells of the nervous system.*

   a. *Meningeal tumours*:

   Type 1. Non-reticulin or collagen forming meningiothelial tumours.

   Var. 1. Cells evenly distributed in sheets; Var. 2. Cells in alveolar arrangement; Var. 3. Spindle cells in interlacing bundles; Var. 4. Combination of varieties 1, 2, and 3.

FIG. 1—Extradural hæmorrhage. Large blood clot is seen under cover of partially reflected duramater (Case 4).

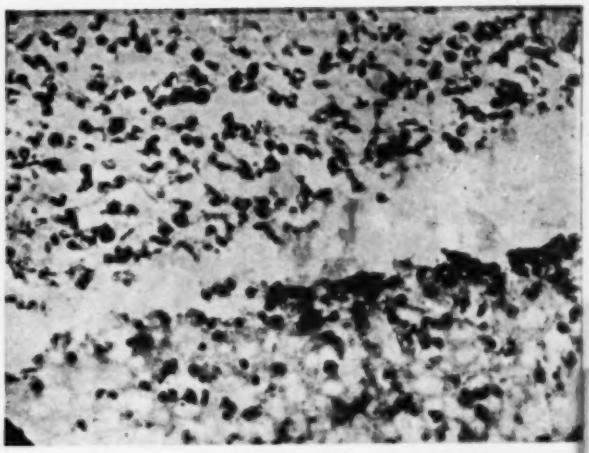

FIG. 3—Traumatic meningitis. Inflammatory exudate of polymorphs seen over the detached meninges of the cerebellum. (Surface layer of cerebellum is only seen). (Case 13). (H. & E. × 200).

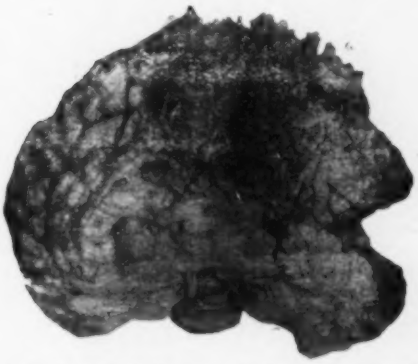

FIG. 4—B. coli infection—ventricular wound, 3 days old.

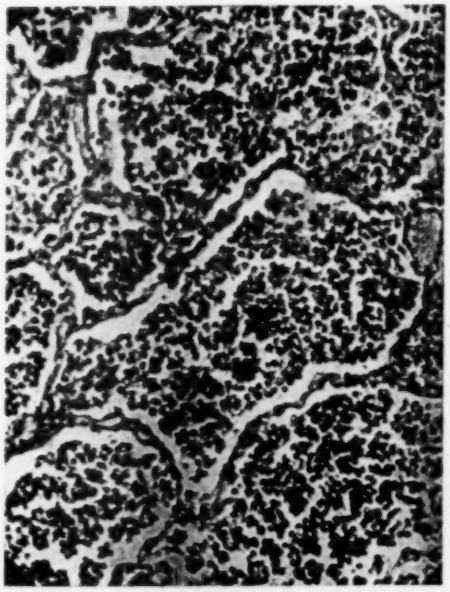

FIG. 2—Bronchopneumonia. The alveoli of lung filled with polymorphonuclear exudate. (Case 11). (H. & E. × 100).

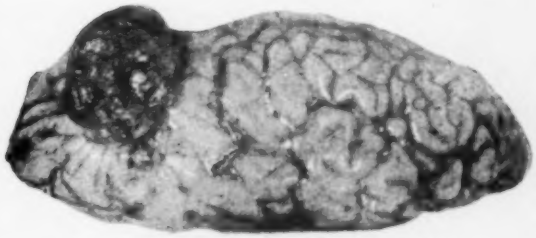

FIG. 5—B. coli meningitis with fungus cerebri.

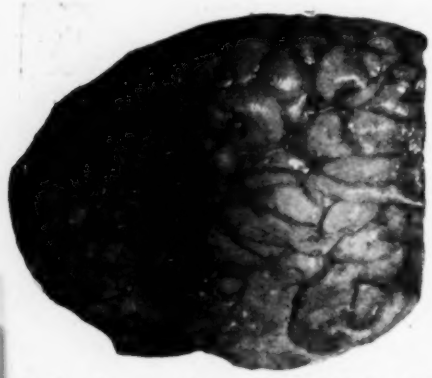

FIG. 6—B. coli—suppurative encephalitis with liquefaction

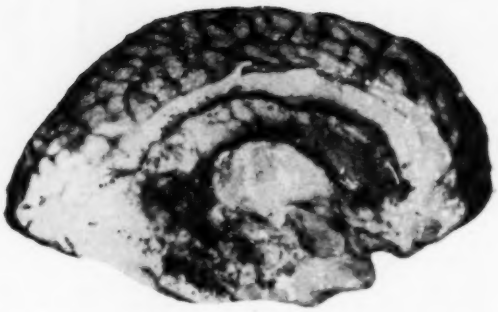

FIG. 8—B. coli—ventriculitis (Note the purpuric spots and areas of softening).

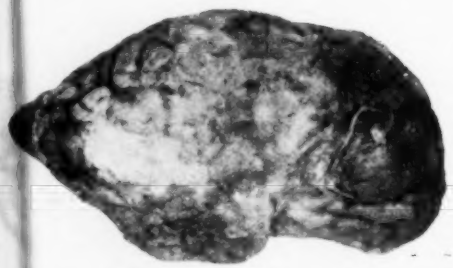

FIG. 7—B. coli—suppurative encephalitis.

FIG. 1—Barium enema in 1948. Shows the filling defect in the rectosigmoid loop.

FIG 2—Barium enema in 1950.

Type 2. Meningothelial tumours of whorl pattern with tendency to form reticulin or collagen.

Var. 1. Variously distributed spindle, round and polyhedral cells; Var. 2. Combination of Type 1, Var. 1 or 2 with whorls; Var. 3. Uniform small whorls; Var. 4. Compact psammoma bodies.

Type 3. Reticulin or collagen-forming fibroblastic tumours of benign type.

Var. 1. Fibrils scanty; Var. 2. Fibrils abundant; Var. 3. Marked fibrosis.

Type 4. Reticulin-forming angioblastic tumours.

Var. 1. Incompletely differentiated with mitoses; Var. 2. Transitional between meningothelial type and angioblastoma; Var. 3. Angioblastoma, capillary or cellular.

Type 5. Non-reticulin or collagen-forming epithelioid tumours.

Var. 1. Cells in columnar arrangement; Var. 2. Roussy and Oberling type.

Type 6. Reticulin or collagen-forming fibroblastic tumours of malignant type (sarcomatous meningiomas).

Var. 1. Spindle cell type; Var. 2. Round cell type.

Type 7. Osteoblastic meningiomas.

Var. 1. Chondroma; Var. 2. Osteochondroma.

Type 8. Lipoblastic meningiomas.

b. *Sheath tumours*:

1. Neurinoma.

3. *Hypophysial Tumours.*

a. *Adenoma*:

1. Eosinophil and 2. Chromophobe.

b. *Craniopharyngioma.*

4. *Dysembryomas.*

1. Teratoid cyst; 2. Pearly tumours; 3. Chordomas; 4. Pinealomas; 5. Papillomas.

5. *Vascular Tumours.*

1. Hæmangioblastoma (Lindau's disease); 2. Angioma racemosum; 3. Capillary telangiectasis.

6. *Metastatic and Invasive Tumours.*

1. Bronchiogenic carcinoma.

7. *Granulomatous Tumours.*

1. Tuberculomas; 2. Syphilomas.

8. *Hæmatomas.*

1. Epidural; 2. Subdural; 3. Subarachnoidal; 4. Intracerebral.

Expansive process of the cranium cause a rise of the intracranial tension and œdema of the brain. The œdema is not proportional to the size of the expansive process. Small expansive processes sometimes cause marked œdema, resulting in neurological signs away from the site of the lesion. Of all the signs indicating the existence of an intracranial tumour, none is so important as papilloedema. Swelling of the eye-grounds is not pathognomonic evidence of a tumour, nor even of increased intracranial pressure, but when present always suggests a tumour, and places the burden of proof upon the diagnosis of lesions of another type. Papilloedema must, of course, be taken as a sign to be correlated with the other neurological findings. It is by no means uncommon to have papilloedema due to intracranial process, when the underlying cause is not a tumour but some other lesion. The pathogenesis of papilloedema associated with increased intracranial pressure is the obstruction to the venous outflow (which is an important factor in the production of papilloedema associated with increased intracranial pressure) and such obstruction is probably due to the pressure on the central vein which traverses the perineural space about the optic nerve. If obstruction to the venous outflow is the only cause of papilloedema it is expected that the entire retina will be œdematous but this is not the usual situation encountered. The suggestion that a colloid chemical factor of unknown nature influences the localization of the œdema to the optic disc and its immediate surrounding may be correct. The increase in pressure caused by a tumour is greatest in the tumour itself, which diminishes in progressively widening circles around it. The rise in intracranial pressure caused by a tumour may be greater in the region of the tumour than in the other parts of the cranial cavity, greater even than in the other parts of the same dural compartments. In most instances the skull acts as a rigid barrier, so that if expansion commences in one compartment, it must occur at the expense of the fluid contents—cerebrospinal fluid or blood—or by displacement towards a region of lesser pressure and ultimately through the foramen magnum. However, the picture of pure centrifugal expansion of the tumour itself is modified by other factors—œdema surrounding the tumour, the effect upon the circulation of cerebrospinal fluid and the presence of the dural partition. Gross œdema may involve the whole of the hemisphere in which a small tumour is situated and this may produce a misleading ventricular picture. When there is blockage of the interventricular foramen, the pressure within the ventricle will increase, and if there is tumour tissue impacted in the foramen this increase may be greater than in the tumour itself. The pressure in the dural compartment in which these conditions exist will be greater than in other regions within the skull. Increase in bulk of the contents of one compartment may result in the herniation of its contents into another and this in turn may aggravate the condition by causing or increasing obstruction to the flow of cerebrospinal fluid. Herniation may occur from side to side under the falx or downwards through the incisura tentorii or the foramen magnum. Transit under the falx will, in the early stages, furnish useful

decompressive effect and it is called the natural decompression but herniation through the incisura tentorii and the foramen magnum is of threatening significance because of the obstruction to the circulation of cerebrospinal fluid and the compression of mid-brain and medulla. The obstruction to the flow of cerebrospinal fluid through the foramen magnum assumes significance after lumbar puncture, as the reduction in pressure below will result in firmer impaction of the tonsils of the cerebellum with production of greater pressure on the vital centres of the medulla. A similar effect may be produced by lumbar puncture in the presence of uncus herniation through incisura tentorii, "lumbar puncture in increased intracranial tension shall undo the natural decompression and the nature ends the organism." Ventricular drainage is indicated in an unconscious patient even in moribund condition due to rise of intracranial tension as one drains a bladder or a gall-bladder; the patient shall become conscious and then one proceeds with localization of the expansive process by pneumography and cerebral angiography to raise a proper osteoplastic flap to deal with the condition surgically.

Localization of Function

During recent years functional methods of localization have done much to alter the maps of the cortex originally differentiated by histological means, although there still remains a correlation between the two. The human cortex is divided into 46 cyto-architectonic areas based on evidence from physiological neuronography and strychninization. Strychnine locally applied acts only when synapses are present on neurons and causes disturbances which are propagated only in the direction of normal conduction—not antidromically. These disturbances appear in records of electrical activity as large sudden voltages which can be recorded at the site of strychninization and from all regions to which the strychninized neurons send axons or collaterals. At the site of the strychninization these sudden transient voltages are many times greater than the ordinary spontaneous activity of the cortex.

Localizing Diagnosis

The localization of certain cerebral functions are taken largely for granted, at least, in so far as physical functions are concerned. Nevertheless, scarcely more than fifty years ago, the subject of localization of any cerebral function was extremely controversial. Even the fact of the contralateral representation of motor functions was then denied by Brown-Séquard. In spite of such opposition the fundamental facts of cerebral localization as outlined in classical lectures of David Ferrier in 1878, were gradually accepted and in 1884 the first real fruits of these labours were born. In that year Bannet first localized, upon neurological manifestations alone, a tumour of the brain which was subsequently removed by Godlee. Since that time the advance has been steady, rapid and it is still continuing.

Harvey Cushing in 1921 first demonstrated that homonymous defects in the visual field are frequently produced by tumours of the temporal lobes. In spite of these studies, our knowledge of function of many areas of the brain are still grossly deficient and errors in localization are frequent. The expansive processes of the cranium cause neurological findings due to œdema of the brain, an increased intracranial tension in addition to the involvement of the local area. The silent areas of the brain do not result in neurological findings, such as area 9, 10, 11, 12, 20, 21, 23, 24, 28, 31, 32, 35, 36, 37, 38, after von Economo and Broadman.

An expansive process situated over the frontal pole on the dominant side (area 10 and 11) may cause aphasia due to the involvement of the Broca's area by the œdema.

An expansive process over parietal lobe (area 7a, 7b) may cause hemiplegia due to the involvement by œdema of area 4a and 4r (motor cortex).

An expansive process in the postcranial fossa causes a bilateral hydrocephalus involving the third ventricle and results in pressure over the chiasma or the little chiasma within the chiasma *i.e.*, the macular decussation giving rise to a central scotoma or bitemporal hemianopsia.

An expansive process of the occipital lobe infiltrating the tentorium and exerting indirect pressure over the paleocerebellum represented by the anterior lobe of the cerebellum, the culmen and the lobulus centralis with clinical menifestations of cerebellar fits as the pressure on the neocerebellum resulting in dysmetria dysdiadokinesis, tremors, positive Romberg's sign, nystagmus.

An expansive process of the frontal lobe of the size of a tangerine may only cause headache and papillœdema.

An expansive process over the right parietal lobe may result in left uncus herniation and pressure on the pyramidal tract in the mesencephalon giving rise to a right hemiplegia showing a deceptive neurological finding of a lesion of the left precentral motor cortex.

An expansive process of the cerebellum may set up false localizing signs, the most common of these is the external rectus paralysis due to the compression of the sixth nerve. The third cranial nerve may similarly be involved although not as frequently. However, if an external rectus palsy or evidence of the involvement of the third nerve occurs in the absence of increased intracranial tension, such palsy may be extremely significant. Bilateral forced or reflex grasping particularly in presence of increased intracranial tension cannot be assumed to be of localizing significance.

An expansive process of the pituitary may result in bitemporal hemianopsia not due to the direct pressure over the chiasma but due to indirect pressure caused by currents in chiasmatic cistern. The vast majority of false localization signs are induced by increased intracranial tension and œdema of the brain. This, gauging the expansive process by the neurological signs alone, was from a surgical standpoint a hit or miss matter at best. During the pneumographic and angiographic diagnosis of cerebral expansive processes, the neurological findings are not taken into consideration.

Case Reports

Case 1—A fiftyfour year old man, came with the following complaints:

Diminution of sensation of smell and taste; pricking sensation of the right arm and leg; unconsciousness after an aura of auditory sounds and periodic shaking of right arm and leg; cramps of the axillary and groin region; head pushed to the left with conjugate deviation to the left, patient during examination had clonic cramps of the right half of the body for few minutes; hyperthermia of 39 degrees; from 31st October headache on the right half, some nausea but no vomiting; shaking of the right side of the body; buzzing in the right ear; incontinence.

The patient had an attack of acute meningitis from 23-8 to 11-9, left facial muscles weaker than the right.

Angiography.—The carotid syphon in its upper part is lifted and straightened, post. cerebral is depressed and straightened. The sylvian group is lifted upward. The expansive process is in the anterior part of the temporal lobe.

Operation.—After raising a right temporal osteoplastic flap, the cerebral surface of the temporal lobe was felt and gave the soft doughy consistency of a tumour, superficial vessels were clamped and undercut and a cutting incision was given over the front part of the temporal lobe and the tumour was exposed. It was thought to be a meningioma, but later found to be a malignant tumour of metastatic origin.

Comments.—Diminished sensation of smell and taste, unconsciousness after aura of auditory sounds were due to the local effects of the tumour. Pricking sensation of the right arm and leg, shaking and cramps of the right arm and leg, conjugate deviation of the head to the left were due to the spread of œdema to the pre- and post-central gyrus, *i.e.*, area 4a, 4r, 1, 2, 3, 5 and area 8 of the precentral motor cortex.

Case 2—A 54 year old man, since autumn 1947 was having balance disturbance, Romberg's sign positive to the left; diminution of hearing and wheezing sounds mostly in the left ear; weakness and shakiness of the right hand, difficulty in holding the pen and for last few months symptoms were stationary; since two months slurring speech; headache of spreading character localized to the back of the head, no change in headache in changing the position of the head.

X-ray appearance of the skull.—Left parietal region has a thinning of the bone and from which there are many tortuous vessels, arteries in a downward direction and veins in upward direction. The foramen spinosum of the left side is dilated, sella turcica is thinned and destroyed.

Operation.—Left parieto-occipital flap was turned down and dura cut around the meningioma, leaving the portion adherent to the tumour. Surrounding vessels of meningioma were cut after applying silver clips and coagulation. Brain covering retracted and the tumour was exposed, later finger removal and cutting the basal vessels after applying silver clips. Blood transfusion 800 c.c.

Comments.—Balance disturbance and positive Romberg's sign were due to high intracranial tension and hydrocephalus. It is a false localizing sign. Diminution of hearing, weakness of the right hand, and slurring speech are due to the spread of the œdema to area 44, 42 of Broadman *i.e.*, the transverse temporal gyri and the central part of the superior temporal gyri, lower part of the precentral motor cortex, area 4 and the Broca's area *i.e.*, area 44. Headache associated with increased intracranial pressure results from traction and displacement of pain-sensitive intracranial structures and is independent of generalised intracranial pressure.

Case 3—A patient, 45 years old, female, came for: headache coming once a month; pain is of acute character spreading to the whole of the head, vomiting; attacks of unconsciousness for one to two hours; acute attacks of pain on the right side of face; diminution of smell since two years, disturbance of taste; corneal reflex diminished on the right side.

Memory disturbance, homonymous quadrantic defect in the superior quadrant with 10/2,000 but with the red object of the same size there is a constriction of the field on the left side and a quadrantic defect on the right side. Dorsum sellæ destroyed on the left side, there is calcification in the parietal and occipital regions.

Angiography.—Anterior cerebral artery is pushed away from the mid-line and the sylvian group are pushed upward, posterior cerebral artery is pushed towards the right side and goes round the calcification in the occipital region. There is an expansive process in the parietal and occipital region.

Ventriculography.—There is a filling defect in the region of the trigonum, occipital horns and upper part of the temporal horns. There is a column of air probably marking the boundaries of the tumour. In antero-posterior view septum pellucidum is displaced to the right side with a filling defect in the body. In all pictures there is hydrocephalus. The calcification is in the area of trigonum, occipital horn and upper part of the temporal horns.

Operation.—Left osteoplastic flap turned down over the trigonum area. There was a big encapsulated tumour situated parasagittally going over the tentorium cerebelli in the region of torcula. As the tumour had infiltrated the lateral sinus, tentorium was cut along the straight sinus and then the tumour traced along the lateral sinus. In order to expose the area of torcula and lateral sinus, the bone was nibbled away behind the transverse sinus and torcula, the sinus was opened in many places which the surgeon sutured. At one place the tumour had infiltrated the sinus and it was blocked by the tumour *i.e.*, at the most extreme end a little medial to the junction of lateral sinus with sigmoid sinus, the sinus was again opened and sutured. Cerebellum was exposed by removal of the tentorium. The tumour was removed in its entirety.

Comments.—Parasagittal meningioma infiltrating the tentorium in the region of torcula has caused very few neurological findings. Headache is produced by traction upon

intracranial pain-sensitive structures such as veins and venous sinuses. There are two types of traction operating in this case, local traction by the tumour mass upon venous sinuses and distant traction by extensive displacement of the brain directly by the tumour and indirectly by ventricular obstruction. Vomiting is due to high intracranial tension, diminution of smell and taste is due to the spread of œdema to the temporal lobe. Attacks of unconsciousness are due to the electronic firing from the hyperexcitable cortex round the tumour and not the tumour itself as it does not give rise to electronic discharge, and involves the diencephalon where the centre of consciousness lies.

Case 4—A male, 50 years old, had the following: diminished memory; attacks of unconsciousness—the first attack on 6-3-48, and the second attack on 7-5-48; psychic changes; uncinate fits, duration 15 minutes at a time after interval of 14 days—fits came daily, sometimes at intervals of one to three weeks; tiredness, aphasia; headache; vertigo; balance disturbances.

Angiography.—The anterior cerebral and pericallosal arteries are dislocated to the right about ½ cm. Carotid syphon is opened out. The sylvian group of vessels are directed upward and medialward. There is a pathological process of the left temporal lobe (malignant glioma).

Operation.—Left temporal osteoplastic flap raised, dura exposed which was very vascular, the bone was thickened and nibbled away. Bleeding points closed by Horsley's wax, dura was snipped and incision was prolonged over a retractor, cautery incision given over the temporal cortex and the meningioma separated by a sucker. It was slowly separated by a retractor and the tumour was dislodged from the base. It was a big-sized meningioma. It was removed by cautery loop piecemeal. The base of the tumour was removed by volsellum forceps, vessels cauterised in the tumour, the middle cerebral artery was passing through the base of the tumour, so part of the tumour was left behind.

Result.—The patient died as the middle cerebral artery was clamped by mistake.

Comments.—Diminished memory, psychic changes, aphasia were due to the spread of the œdema to the frontal lobe of the dominant side.

Uncinate fits were due to the spread of œdema to the rhinencephalon. Vertigo and balance disturbance were due to high intracranial tension. Attacks of unconsciousness were due to hyperexcitable focus in the cortex round the tumour sending electronic firing involving the diencephalon.

Case 5—Female, 29 years old, has left-sided facial paresis for the last ten years and complains of the following: buzzing sensation in the left ear; weakness of extremities—first legs, then arm on the left side, later on the right side; there was a band of anæsthesia over the face and from one month anæsthesia more marked; diplopia, visual hallucinations; memory disturbances; weakness of sexual function; increase of weight; loss of hair; from 1st January the patient had attacks of stiffness with pain over the arm, leg, neck and so she had to remain in bed.

Ventriculography.—The frontal horns are separated apart showing tumour of the septum pellucidum.

Operation—Cushing's temporal decompressions. A longitudinal incision was given above the ear on the right side in the temporal region instead of the usual vertical one. Burr hole was made and the bone was nibbled away. Dura was not incised, but only the outer layer of dura was removed to avoid a protusion. A layer of dura was left behind to avoid pressure on the brain stem. Temporal muscle and fascia sutured.

Comments.—Some of the central tumours go on for ten years. Facial paralysis had nothing to do with the tumour. Trigeminal and abducent involvements were due to high intracranial pressure. There were no symptoms of thalamus involvement. The tumour was in the septum pellucidum causing compression symptoms. Weakness of extremities was due to the spread of œdema to the precentral motor cortex. Memory disturbance was due to the spread of œdema to the frontal lobe. Weakness of sexual function, increase of weight, loss of hair, were due to internal hydrocephalus involving the third ventricle causing a pituitary syndrome.

Case 6—A man, 37 years of age, had epileptic attacks since his childhood. He used to have 15 attacks a day with unconsciousness, cramps, involuntary passage of urine and fæces. At the age of 12 years there was a quiet period of 1½ years. Then the attacks came spontaneously, no vomiting or headache. X-ray of skull showed no pathological or vascular changes of the bottom of the skull, no destruction. There was a little pineal calcification which was not displaced. In the middle cranial fossa 1½ cm. behind sella turcica there was regular calcification.

Encephalography.—No displacement of the ventricular system, no dilatation or local deformity of frontal horn or 3rd ventricle. Both temporal horns were lying in the usual place. In the middle and front portions was found calcification with concavity on the lateral side of the temporal horn which was also wide.

Angiography.—Sylvian group of vessels were pushed upwards and medially. Fundus, no pathological changes.

Operation.—Right temporal osteoplastic flap turned down, middle temporal gyri had a soft yellow appearence, cautery cut, there was a cystic tumour which was separated round about and removed. It was diagnosed as a case of oligodendroglioma.

Comments.—Epileptic attacks were due to a hyperexcitable focus in the cortex round about the tumour setting up an electronic firing involving the 35,000 Betz's cells (giganto-pyramidal cells) on each side in precentral motor cortex in area 4a, 4r, sending a volley of impulses by corticospinal tract to anterior horn cells giving rise to convulsions. The area 4s in the precentral motor cortex has pyramidal cells and is called the suppressor strip to moderate the hyperexcitable cells of area 4a and 4r and axons from pyramidal cells of 4s run to the formatio reticularis of medulla and in the lateral columns of spinal cord to moderate the electronic firing from the anterior horn cells. Let me propagate a new theory. All patients with convulsions would have broken their arms and legs if nature had not provided the suppressor strip to moderate the hyperexcitable 35,000 Betz's cells of each motor cortex.

Acknowledgment

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OBSERVATIONS ON THE MECHANISM OF HAEMOLYSIS IN BLACKWATER FEVER

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It is generally recognised that blackwater fever is somehow or other related to malarial infection. Residence in hyperendemic areas, repeated attacks of malarial fever, hæmolytic strains of parasites and administration of antimalarial drugs, specially quinine, have all been suggested as factors in the development of the syndrome. But the actual mechanism of the sudden hæmolytic crises is obscure. Maegraith (1948) has collected all the up-to-date works on the subject in his admirable and comprehensive monograph which should be consulted by everyone interested in malaria and blackwater fever. Foy and his co-workers (1945) observed rapid dissolution of red blood cells of a normal person when introduced into an actively hæmolysing case of blackwater fever and also found that the red cells from an active case of blackwater fever when transfused into a normal person undergo the same change. This observation is very illuminating. It suggests that the red blood cells in blackwater fever are abnormally susceptible to the normal lytic processes in the body while the plasma contains an abnormal lytic substance as well. This indicates that defects both in the red cells and in the plasma contribute to the hæmolytic process in this disease. As regards the red blood cells, osmotic and morphological changes have been described by various workers to confirm this lytic tendency in this disorder. But reports on this item are conflicting. Thus some workers have recorded increased fragility to hypotonic saline (see Maegraith, 1948), on the contrary others have reported normal or decreased fragility (Dudgeon 1920; Ross, 1928). Foy and Kondi (1943) have observed increased fragility to lysolecithin although saline fragility was normal.

Some workers have described spherocytosis of red blood cells (Gear, 1946). Foy and Kondi (*loc. cit.*) have also recorded decrease in diameter-thickness ratio of erythrocytes which thus appear to be swollen up. This indicates some alteration in the cell membrane with loss of elasticity. Considerable speculation has been raised on the cause and significance of this observation in blackwater fever and malaria. Thus spherocytosis has been taken to mean a damaged cell-membrane (Foy and Kondi, 1943) through which hæmoglobin can easily leak out. The swollen cells have been believed to face resistance in the smaller blood vessels of internal organs and so undergo phagocytosis (Whipple, 1941). Malarial anæmia and slowing of splenic circulation have been thought (Vint, 1941) to facilitate overaction of the stabilisation phenomenon of Fahracus (1939) and thus promote spherocytosis which means increased tendency to hæmolysis. Gear (1946), on the other hand, considers spherocytosis as a manifestation of the action of hæmolysin on the cells.

Various changes in the plasma have been thought at least to contribute to the hæmolysis observed in blackwater fever. It is known that protein and cholesterol in normal plasma inhibit the hæmolysis induced by saponin, bile salts and by extracts of normal tissues. Maegraith and his co-workers (1943) collected evidence from blackwater fever cases to

suggest that the protective effect of plasma proteins and cholesterol is reduced in blackwater fever.

The possible relation of red cell destruction to some form of antigen-antibody reaction has been suggested by several workers. Such a possibility finds support in the clinical histories of blackwater fever cases that they are subjects of repeated malarial attacks. Presence of pronounced erythrophagocytosis in chronic malaria (Taliaferro and Mulligan, 1937; Yorke *et al.*, 1929; Thompson, 1924; Coggeshall, 1937) strongly suggests the possible role of an opsonic type of antibody, but it is difficult to believe that the rapid hæmolysis of blackwater fever can be caused by such an erythrophagocytic phenomenon. The occurrence of auto-agglutination of erythrocytes in malaria and blackwater fever has led Oliver-Gonzales (1944) to suggest that the malarial parasite carries some antigen related to human isoagglutinins. Cold agglutinins have been demonstrated in the serum of blackwater fever cases (Oliver-Gonzales) but its significance has not been properly evaluated. Gear (1946) has advanced an interesting hypothesis of auto-antigen and auto-antibody being concerned in the hæmolysis of blackwater fever. He believes that organs or tissues of a particular species of animal after being acted on by an invading parasite or its products, behave as antigen when introduced into another animal of the same species. It gives rise to what may be considered as a homologous antibody. Gear extends this idea and thinks that in blackwater fever, the parasite repeatedly acting on the red blood cells renders the latter antigenic within the patient himself; eventually the corresponding antibody (hæmolysin, autohæmolysin or autolysin) is formed and is stored in the reticulo-endothelial system, particularly in the spleen and when this store of hæmolysin finds an outlet into the general circulation, destruction of erythrocytes, hæmoglobinæmia and hæmoglobinuria give rise to the syndrome of blackwater fever. Gear's theory seems very attractive and his scheme can be tabulated as follows:—

(*i*) Erythrocyte + malarial parasite and/or quinine → autoantigen.
(*ii*) Autoantigen → autoantibody (hæmolysin, autohæmolysin or autolysin).
(*iii*) Erythrocyte + autolysin → sensitised red cells.
(*iv*) Sensitised red cell + complement → hæmolysis.

It may be observed that complement is used up in the process of hæmolysis postulated by Gear.

In the face of so much confusion prevailing around the mechanism of hæmolysis in blackwater fever, we were tempted to carry out a few investigations with the red blood cells and with the serum of a case of blackwater fever admitted under one of us (N.R.K.).

Case Report

D.N.B. male, aged 30 years, a resident of Krishnanagar and a daily passenger to Calcutta, was admitted at 10-30 a.m. on 8-12-50 as a case of blackwater fever. The patient gave history of frequent attacks of malaria in the past and of an attack of blackwater fever 9 years ago. For the last 8 days before his admission he had suffered from low irregular fever. On 8-12-50 at about 7 a.m. he took a tablet of quinine sulphate (5 gr.). In the train he had slight pain in the abdomen and passed urine. It was of dark red colour and he knew that he had blackwater fever. He went to a friend's place in Calcutta and passed red urine. He walked to the hospital—a distance of about a mile and passed similar urine in the emergency room. His general condition was good but he was apprehensive and repeatedly enquired if he would live.

On Examination the following were found:

Temperature—101·4°F, pulse—90 p.m., respiration—26 p.m., blood pressure—120/60 m.m.Hg., anæmia—moderate, jaundice—slight, œdema or cyanosis—nil, tongue—pale and dry, spleen—hard and enlarged 2 fingers' breadth below the costal margin, liver—not palpable, no other abnormality was found.

Blood—showed hæmoglogin 75 per cent, total W.B.C. 9,500 per cmm., neutrophils 88 per cent, lymphocytes 12 per cent, monocytes—nil, eosinophils—nil, malarial parasites—none found.

Urine—was of reddish colour, reaction—acid, sp. gravity —1,006, albumin—present in large amount, sugar—nil., test for blood—strongly positive, microscopically no R.B.C., no pus cells, no casts or crystals seen.

Stool—showed no abnormality.

Blood on 9-12-50 showed hæmoglobin 57 per cent, bilirubin content.—3.5 mgm. per cent, van den Bergh reaction—immediate direct positive, N.P.N.—58 mgm. per cent, cholesterol—125 mgm. per cent.

Clinical course—The patient was put on absolute rest and was given an alkaline mixture containing 30 grs. each of sodium bicarbonate and sodium citrate 6 hourly, vitamin C 100 mgm. and paludrine 0·1 gm. thrice daily. At about 6 p.m. on 8-12-50 he had a rigor and the temperature went upto 104·8°F. He passed a large amount of urine of port-wine colour. On the next day the temperature varied between 102° and 103·8°F., the blood pressure was 98/56 mm. Hg. and hæmoglobin was 57 per cent (Sahli). 300 c.c. of appropriate blood of 'O' group was transfused. The hæmoglobinuria persisted and the patient became restless. On 10-12-50 the blood pressure was 100/45 mm.Hg. and hæmoglobin 40 per cent. Paludrine was discontinued. He had an antihistaminic drug (diatrin) 50 mgm. thrice daily. Adrenal cortical extract, glucose, vitamin C, nikethamide and sodium acetate were given parenterally and 340 c.c. of blood of 'O' group was transfused. On 11-12-50, his general condition was low, the temperature varied between 101°F and 104·8°F., blood pressure was 95/45 mm.Hg. and hæmoglobin 30 per cent. He had another transfusion of 240 c.c. of blood of 'O' group. On 12-12-50 his condition became worse and the patient became comatose. There was very little secretion of urine which was, however, of almost normal colour. The temperature gradually came down and the pulse became quicker and very feeble. The hæmoglobin came down to 20 per cent. Another 150 c.c. of blood of 'O' group was transfused at about 1 p.m. The condition gradually deteriorated and the patient expired at 11 p.m.

Material—Blood was obtained from the anterior cubital vein of the patient on the third day (10-12-50) of his illness. About 10 c.c. of blood was taken in a double oxalate tube and another 20 c.c. in a dry sterile tube for collection of

serum. The finger of the patient was pricked for estimation of hæmoglobin and for erythrocyte count. The venous samples showed gross evidence of hæmoglobinæmia.

Methods—The following examinations were done:

(1) To detect alteration in the red blood cells—(*a*) Total red cell (*b*) hæmoglobin, (*c*) packed cell volume, (*d*) mean corpuscular volume, (*e*) mean red cell diameter (average of 500 cells), (*f*) mean corpuscular average thickness, (*g*) fragility of the erythrocytes washed in normal saline to hypotonic saline and to mechanical trauma.

(2) To demonstrate any possible evidence of antigen-antibody reaction in the serum—

(*a*) Acid cold hæmolysin (Dacie, 1950 b), (*b*) Cold agglutinin after the method recommended by Humphrey (1947). (*c*) Donath-Landsteiner reaction. (*d*) Determination of complement titre in the serum.

A sample of blood was also collected from one of us. 5 c.c. was placed in a double oxalate tube and 5 c.c. in a dry test tube. The cells and the serum from these samples served as control in the tests for saline fragility, mechanical trauma, Donath-Landsteiner reaction and determination of complement titre.

Results—Results are tabulated as under:

Examination	*Results*	
Hæmoglobin	40% or 5·8 gm. (Sahli-Adams)	
Total R.B.C.	1·69 million/cmm.	
Packed cell volume	13%	
Mean corpuscular volume ...	76·9 cμ	
Mean cell diameter	7·02 μ	
Coefficient of variation (*v*) ...	8·8%	
Standard deviation	0·62 μ	
Mean corpuscular average thickness ...	1·98 μ	
M.C.D. : M.C.A.T.	3·5 : 1	
Malarial parasites	none found	
Fragility of corpuscles:		
(*a*) Mechanical trauma* ...	nil	
(*b*) Osmotic saline fragility† ...	*Patient*	*Control*
Initial	0·5 %	0·52%
Complete	0·36%	0·34%
Mean corpuscular fragility	0·42%	0·4 %
Acid cold hæmolysin ...	Negative.	
Cold agglutinin	Positive upto 1 in 250 dilution at 2°-4°C.	
Complement titre	1 in 4 : Control—1 in 15.	
Donath-Landsteiner reaction ...	Negative.	
Direct Coomb's test	Negative.	

Discussion

Changes in red blood cells.—The red cells show microcytosis (M.C.V.—76.9cμ). This observation differs from those of Yorke and his colleagues (1929) and of Fairley and his co-workers (1938) who found the mean corpuscular volume unchanged in blackwater fever. Gear (1946), however, observed microcytosis in his two cases of blackwater fever, which also showed hyperchromicity of the erythrocytes. This feature—microcytosis together with hyperchromicity was taken by him as evidence of spherocytosis. We have not tried to estimate the mean corpuscular hæmoglobin due to the presence of hæmoglobinæmia, but calculation of diameter thickness ratio (3·5:1) indicates little evidence of spherocytosis of the red blood cells when we consider that the normal range of this ratio falls between 4:1 to 2:1 (Haden, 1935). Spherocytosis of the red blood cells has been described in blackwater fever (Foy and Kondi, 1943) and as indicated before, chiefly on the basis of this observation numerous hypotheses have been advanced to explain the hæmolysis of blackwater fever. Absence of spherocytosis in our case suggests that this erythrotic change is not constant in blackwater fever and cannot, therefore, be considered to take any major part in the mechanism of hæmolysis.

The fragility of the red blood cells to hypotonic saline or to mechanical trauma did not show any significant change which could in any way be held responsible for the hæmolysis. It is generally agreed that spherocytes are more fragile to hypotonic saline and to mechanical trauma (Dacie, 1950 a) and absence of any significant change in fragility in the present case supports our observation on spherocytosis made from direct measurement of cell size.

The question of antibody.—Donath-Landsteiner reaction with and without previous chilling was negative. This appearently indicates that there is no gross antibody present in the serum in the form of hæmolysin which can account for the hæmolysis observed in blackwater fever. But this does not exclude the possibility of antigen-antibody reaction entering into the mechanism of hæmolysis especially when we consider the fact that cold agglutinin was demonstrable to a significant titre (1 in 250) in the serum. It has come to light in more recent years (Dacie, 1950 b) that cold agglutinin is associated with certain hæmolytic anæmias and with Raynaud's disease with or without hæmoglobinæmia and also with virus pneumonia in which disease episodes of acute hæmolysis have sometimes been observed (Finland *et al.*, 1946; Colmers and Snaveley, 1947). This agglutination in the cold *in vitro* may in some way or other be related to hæmolysis *in vivo*. Stats and his co-workers (Stats, 1945; Stats and Wassermann, 1943; Stats, Wassermann and Rosenthal, 1948) have shown that such agglutinated corpuscles undergo lysis if kept in the cold and tapped repeatedly. Dacie (1950 b) has found that cold agglutinin may often be associated with a hæmolysin which is demonstrable only after cold incubation in an acid medium. Such an acid cold hæmolysin could not be found in our case of blackwater fever, nor were the agglutinated corpuscles lysed on tapping, but it may be observed that the titres of cold agglutinin in the cases of Dacie and of Stats and his colleagues were very high. In the light of

* The red cells washed and suspended in normal saline and subjected to shaking in a Kahn shaker for half an hour together with a control sample from a normal person did not show any trace of hæmolysis.

† Hæmolysis starts later and is completed earlier. The mean fragility is very slightly increased as also the lower limit. On the whole the change in fragility does not appear to be significant.

demonstration of high agglutinin titre (in thousands) by these workers, the significance of such low titre as found in our case appears to be lessened but Dacie (1950 b) has suggested that the titre of cold agglutinin cannot necessarily be correlated with the presence or degree of hæmolysis or hæmoglobinuria *in vivo*. The relevant point that emerges out of these observations is that an antigen-antibody reaction almost similar to that of the above workers may be concerned in the mechanism of hæmolysis. In our case we could not exclude any previous attack of virus pneumonia which might account for the presence of cold agglutinin but the demonstration of the same antibody by Oliver-Gonzales (1944) would seem to emphasise its significance in blackwater fever. In the cases described by Dacie (1950 b) cold agglutinin in the serum was associated with the presence of incomplete antibody. Demonstration of such antibody in blackwater fever cases would be very helpful in confirming that hæmolysis in this disease is related to antigen-antibody reaction. In our case, direct Coomb's test proved negative which means no antibody (human-globulin) remains absorbed to the red blood cells.

The presence of cold agglutinin seems also to be of some significance in relation to the hypothesis of Gear (1946) indicated above. Cold agglutinin in trypanosomiasis, another protozoal disease, has been regarded as an 'auto-antibody' resulting from 'auto-immunization' by a substance formed by the action of protozoal protein on human tissues (Humphrey, 1947). A similar state of affairs may justifiably be expected to exist in blackwater fever which is universally believed to be associated with infection by a sister protozoon. We have also found a very low titre of complement (1 in 4) in the serum from our case of blackwater fever. While admitting that there may be many other causes for the presence of such low titre of complement, it is tempting to think that Gear's autolysin has used up the major part of the patient's complement in precipitating the episodes of hæmolysis, hæmoglobinæmia and hæmoglobinuria.

The progressive development of anæmia in the present case in spite of daily transfusion of blood for four successive days suggests that the donor's cells could not make any impression. The work of Foy and his co-workers (1945) on the fate of transfused normal red cells into the circulation of a blackwater fever case indicates that the donor's cells met with the same fate as our patient's own red blood cells when they came in contact with the circulating hæmolysin. This fact seems to raise serious doubt about the utility of transfusion of blood during an attack of blackwater fever, hæmolysis of the transfused cells may be thought only to aggravate the course of the disease. But when the process of hæmolysis in blackwater fever is considered as a manifestation of an antigen-antibody reaction, transfusion of blood appears to be definitely beneficial as the transfused cells will at least block some of the circulating antibody.

Summary and Conclusions

A case of blackwater fever has been described. An attempt has been made to find out any alteration in the morphology and in the fragility of erythrocytes or any serological change which might throw some light on the mechanism of hæmolysis in this disease. There is no evidence of any red cell change which can account for the hæmolysis. On the other hand, demonstration of cold agglutinin in the serum is taken to support the idea that an (auto) antigen-antibody reaction is concerned in the mechanism of hæmolysis, although no warm, cold or acid cold hæmolysin could be detected.

Acknowledgment

We wish to express our thanks to Dr. A. K. Dutta Gupta, Superintendent, Nilratan Sarkar Medical College Hospitals, for permission to publish the case and to Dr. A. Sen, Director, Blood Bank, Medical College Hospitals, Calcutta, for doing the Coomb's test for us.

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DIAGNOSIS AND TREATMENT OF ABDOMINAL TUBERCULOSIS

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Hubli

There are three distinctive features of tuberculosis in general and abdominal tuberculosis in particular, *viz.*,

1. Tuberculosis in its earlier stages mimics many common ailments.
2. Wherever the prominent clinical manifestations may be, in a particular case of tuberculosis, the infection has to be considered as a general disease and treated as such, besides treatment of the local condition.
3. Symptomatic relief, which the patient gets after some treatment, does not mean a cure and the treatment has to be continued for a very long period.

To avoid repetition, I shall restrict to intestinal, peritoneal and glandular (mesenteric) tuberculosis, from the clinical and therapeutic aspects only.

Intestinal Tuberculosis

The varieties of intestinal tuberculosis, depending upon the condition it resembles are:—(1) typhoid form, (2) dysenteric form, (3) obstructive form, (4) appendicular form, and (5) pelvic form.

(1) *Typhoid form.*—Though uncommon, it is the most severe one. It mimics typhoid fever in the beginning, except that the pulse rate may be in keeping with the temperature or be faster from the beginning. A peculiar symptom often noticed and to which I attach a great clinical significance is a sensation of burning all over the body, especially the soles of feet and the back. I have seen a few cases only of this type and so am unable to evaluate the significance of this symptom. Fever continues and one patiently waits for the natural remission to occur. Diarrhœa is a marked feature. At the end of three weeks, when there is no progress as expected, one starts doubting his diagnosis and, for the first time, begins to think of the infection being possibly tuberculosis. Patient's condition is by now very low. Respiratory system is usually involved. He looks cachectic. Laboratory investigations, if possible, are now done. Negative Widal test and sputum showing acid-fast bacilli confirm the diagnosis. Skiagram shows more involvement of the lungs compared to the clinical findings. Peritoneal effusion may also be added to the picture and the patient's condition causes anxiety. Here, prognosis is not very good and even in favourable cases it means a prolonged confinement to bed. In incurable cases, the terminal event is involvement of the central nervous system.

(2) *Dysenteric type.*—It is seen in young adults. I think that this is the commonest variety of intestinal tuberculosis met with in this part of the country. It is a secondary infection from lung affection, which may, however, be quiescent at the time of detection of the intestinal tuberculosis. Whether the intestinal infection is by ingestion or *via* blood stream, is difficult to decide. Probably it is through both.

In the early stages the patient complains of vague symptoms like anorexia and occasional diarrhœa. He often neglects these. After a time, he begins to feel tired in the evening and feeling feverish, is inclined to lie down after the day's work. He consumes less and less amount of food. Attacks of diarrhœa become more frequent and prolonged. He then consults the doctor. With astringent mixtures and diet restrictions, the patient improves for the time being, but he returns shortly afterwards. One then notices a fast pulse rate, a small rise of evening temperature and tenderness with occasional gurgling and some distension of the lower abdomen. Tongue may be slightly reddish at the borders. Lungs may or may not be found involved. The doctor probably advises a course of emetine injections and a strict dietetic regime and rest. Patient again feels better but he returns after an interval with a relapse of his trouble. Abdominal pain increases, becomes more persistent, and after a time the abdomen is felt as typically "doughy." Then one suspects intestinal tuberculosis, and examination of stools, skiagram of the chest and gastro-intestinal tract, establish the nature of the lesion.

The three important features are: (*i*) evening rise of temperature (*ii*) absence of blood and mucus in the stools and (*iii*) relatively mild griping compared with frequency of stools.

(3) *Obstructive type.*—It is commonly seen. I have not noticed the lesion being more common in females as Dr. Gharpure states.

It starts insidiously with uneasy feeling in the right iliac fossa and with anorexia and constipation. Laxatives or purgatives are resorted to by the patient, but the improvement is little and short-lived. Uneasiness then becomes mild pain which gets worse with purgatives. Anorexia and lassitude increase. The physician notices tenderness and fullness and also a palpable lump in the right iliac fossa. Barium meal and enema skiagrams reveal (*i*) stasis at the ileo-cæcal region (*ii*) incomplete filling or distortion of the cæcal shadow. Here, x-ray helps to establish the diagnosis of ileo-cæcal tuberculosis and differentiates it from chronic appendicitis. Appendix has often been wrongly blamed in some such cases and removed, of course, with no benefit to the patient.

If allowed to progress, it may lead to acute intestinal obstruction needing an emergency operation

which is definitely more risky than a well-planned short circuit operation earlier.

(4) *Appendicular type.*—I mean thereby an acute or subacute appendicitis caused by M. tuberculosis. Inflammation may be primarily confined to the appendix or may have been spread from the peritoneum or ileum. Signs and symptoms resemble acute appendicitis except for absence of leucocytosis.

(5) *Pelvic type.*—This is usually seen in females during the child-bearing period. She complains of heaviness in the lower abdomen, amenorrhœa, loss of weight, anorexia and weakness. On examination, a mass, usually on one side in the pelvis, is felt. It may be globular or irregular, may be firm or soft, may be slightly movable or fixed. This mass is formed by some coils of ileum adherent to each other and also to the uterine adnexa on one or both sides. The contour and consistency of the mass depends upon fibrosis present. The round mass and amenorrhœa are likely to lead to the diagnosis of an ovarian cyst. I believe, the initial lesion is in one of the fallopian tubes, subsequently spreading to peritoneum, intestines etc.

Peritoneal Tuberculosis

This, except in miliary tuberculosis, is secondary to intestinal and tubal infection. In fibrous peritonitis fibrosis is a prominent feature, while in the other ascitic form, there is effusion in the peritoneal cavity, differentiation from other causes of ascites being mostly a process of exclusion only.

Glandular (Mesenteric) Tuberculosis

Secondary involvement of these glands in primary intestinal tuberculosis is not included here. A few cases of primary major involvement of these glands seen by me, had big fixed masses in the abdomen. Severe cachexia follows obstruction to intestinal lymphatic outflow.

Although grouped separately, mixed types may occur.

I shall here mention two cases of rarer varieties, seen by me:

(*i*) In a child of six years, while operating for inguinal hernia, the sac contained omentum, studded with tubercles, though with no symptoms of this disease.

(*ii*) A patient who subsequently died of meningeal tuberculosis, showed a tubercular stricture of sigmoid colon with tubercular ulcerations of the mucosa, but with no lesion on the peritoneal aspect. This was in pre-streptomycin days.

Treatment

As said in the beginning, abdominal tuberculosis has to be considered as a general infection. Treatment, therefore has to be on general lines besides local treatment, if any. These are:

(*a*) *Rest*—It should be not only adequate but complete. This very important part is usually neglected by the patient, as long as he is able to move out.

(*b*) *Food*—It should be easily assimilable and one leaving only a little residue. Milk should be the mainstay and everything else is supplementary. Six to eight cups of milk per day, a small quantity at one time, is what I insist on my patients. If he feels hungry, he is permitted to have other suitable food besides milk.

(*c*) *Vitamins*—I insist on a combination of vitamins the multivitamins, in adequate doses.

(*d*) *Medicinal treatment*—Symptomatic treatment with sedatives, astringents and expectorants is given as required.

(*e*) *Streptomycin, P.A.S. and Surgery*:—It seems, there is an impression that streptomycin is all in all in the treatment for tuberculosis and that surgery has no place in it. In my opinion, the situation is exactly the reverse. Streptomycin has made surgery possible in more cases than before. These drugs are definitely indicated in acute febrile cases and also as post-operative treatment.

Surgery is definitely indicated in the following cases:—

(1) Obstructive type—may be in adhesive type or in hypertrophic ileo-cæcal variety.

(2) Obstructive type of tubercular appendicitis.

(3) Ascitic type where effusion does not subside with usual treatment.

(4) Cases with intestinal fistulæ, which do not respond to medical treatment in a reasonable period.

Surgical treatment has either to be radical or palliative, as is suitable in individual cases.

I, as a rule, use streptomycin and P.A.S. combined, as the two act synergistically, and it is believed that P.A.S. delays development of streptomycin-resistant strains. Only when patients cannot tolerate, they are advised to use the drugs singly. Such occasions have been few after the availability of dihydrostreptomycin.

I do not give more than 20 gms. of streptomycin at a stretch. After a gap of three weeks, the course is repeated, the usual dose being ½ gm. twice a day.

In suitable cases, streptomycin and P.A.S. definitely improve the general condition of the patient, clinical improvement being proportionately more marked than improvement in the pathological process.

I think, streptomycin has considerable toxic effect on the myocardium. In pulmonary tuberculosis

x-rayed at intervals, I have definitely noted the enlargement of the cardiac shadow. As I have no facilities for the E.C.G. studies, I cannot say definitely whether it means actual damage to the myocardium.

I shall describe two cases illustrating my remarks made previously about the disease:

Case Reports

Case 1—A young Hindu aged about 25 years, given to outdoor healthy life, in farming, complained of vague abdominal pains with fever and diarrhœa. *On examination*, I noticed generalised tenderness in the lower abdomen with a little bulging and temperature 100°·8 F. I learnt that this patient had two similar attacks previously, which subsided without any treatment. He never had passed blood or mucus in stools. Lungs revealed signs of bronchitis, and the patient brought up a little sputum occasionally. He was treated with a few emetine injections and bismuth mixture before I saw him. His stools showed no amœbic infection, his sputum no Koch's bacilli. He was put on milk diet and absolute rest. Lungs on x-ray showed old *doubtful* tuberculous lesions. On the provisional diagnosis of the case as one of intestinal tuberculosis, I treated him with streptomycin and P.A.S. He made a rapid recovery and by the end of the week, he was free from symptoms. Thereafter he was allowed a little food. After 20 gms. of streptomycin, he felt very much better. After three weeks, he was given another course of 20 gms. I thought that now he could move about and so he was sent to a village in the open air, all the time continuing with P.A.S. and multivitamin tablets. He returned after a month and within a week of his return, he developed acute spasmodic pain in the right iliac fossa, with vomiting. There was no fever and pulse rate was normal. I thought that I was wrong in my diagnosis and that the patient was suffering from chronic appendicitis, which had then assumed an acute obstructive form. He was immediately operated upon and I found that I was quite right in my first diagnosis. The parietal peritoneum, at the site of the paramedian incision, was ¼" in thickness and was studded with tubercles. Terminal coils of ileum were also studded with tubercles. Cæcum was completely free. Appendix was congested and thickened to the size of a little finger with a constricting ring, about ½" from the base, with the surface showing multiple tubercles. To remove the appendix or not was the issue. Being convinced that it was an obstructive type, I removed the appendix and closed the abdomen. Streptomycin was given postoperatively. The patient made an uneventful recovery and is well upto this day.

My point in mentioning this case, is that in spite of having had 40 gms. of streptomycin before operation and apparent clinical recovery, the pathological condition noted at the operation, was extensive.

Case 2—A Muslim railway worker, middle aged, was seen by me about five years ago. From signs and symptoms he was diagnosed as a case of hypertrophic type of ileo-cæcal tuberculosis He was asked to undergo x-ray investigation and be operated upon, if necessary. I lost sight of the patient only to see him again six months later with severe abdominal pain and diarrhœa. During the interval, he was treated outside with streptomycin and had just finished the course. I now thought that this patient had developed ulcerative lesions superimposed on the original condition. I admitted him into the hospital and started giving him streptomycin and P.A.S. He developed an allergy to streptomycin, as a result he had to be kept on P.A.S. only. The patient became afebrile and his diarrhœa improved. To his bad luck, he was now constipated and started getting pain with distension of the lower abdomen with visible peristalsis. His condition being low, even a short circuit operation was thought to be dangerous. He was kept on P.A.S. and I.V. infusions. He gradually improved and his radiograms showed improvement in his intestinal condition. His condition having improved, ileocolostomy was done. He is now free from abdominal pain and constipation.

My idea in mentioning this case, is to show that surgery was made possible, largely due to streptomycin and P.A.S.

THE MECHANISM OF HEADACHE

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Disergark

Most of the recent work in connection with headache has been concerned with its mechanism though this is not yet properly understood. The works of Pickering and Wolff have, however, added much to our knowledge in this direction. Certain basic facts regarding conception of headache may be noted at the outset. There is no headache centre in the brain. The brain tissue is as such not sensitive to pain though it is through cerebral function that the pain comes into consciousness. All painful phenomena with the single exception of pain arising spontaneously from thalamus as a result of appropriate lesions, result from some sort of peripheral stimulation which for one reason or other, owing to repetition, intensity or other essential characteristics give rise to impulses which are appreciated as pain or headache. The following structures at the head level have been found to be pain-sensitive:

(1) Investing tissues outside the skull.
(2) The basal dura with its tentorial shelf.
(3) The blood vessels.
(4) Certain nerves that carry pain to consciousness—these are sensory branches of 5th, 9th and 10th cranial nerves and upper three cervical nerves.

There are certain other structures at the head level which are definitely not sensitive to pain. These are—

(1) The calvaria (without the periosteum).
(2) The brain tissue.
(3) The pia, arachnoid and most of the dura.
(4) The ependymal lining of the ventricles and the choroidal plexus.

Blood vessels at the head level.—The recent works have emphasized the important role of blood vessels in the causation of headache. They are the extracranial arteries such as temporal, the dural arteries such as middle meningeal, and the cerebral arteries and great venous sinuses with their tributaries. These vessels have been found to be highly sensitive to distension, traction and pressure. All vessels in the body are to some extent sensitive, but the cranial arteries are particularly so. A reference may be made at this stage to the speciality of cerebral arteries. Anatomically these arteries have thinner walls, more collapsible and presumably more distensible than arteries elsewhere. Physiologically they are concerned in supplying the structures which are most susceptible to oxygen deprivation. The pathological change of arteriosclerosis is more commonly seen in these arteries than others of comparable size with possible exception of coronaries. In the pharmacological field it is quite likely for these vessels to react exceptionally to chemicals produced in the body or administered therapeutically or otherwise.

The present trend of opinion is towards recognition of vascular basis in most headaches, the usual mechanism in the majority of cases being dilatation, distension annd traction of the cranial arteries. Rarer causes are displacement of venous sinuses, irritation and traction of the pain-sensitive nerves and structures etc.

The throbbing character of certain types of headaches has always been presumed to be vascular in origin. Evidences in favour of this presumption have been afforded in recent years by the pharmacodynamic studies of headache-producing or alleviating effects of certain drugs and also by direct inspection of blood vessels before, during and after occurrence of the headache.

If headache is to be explained mostly on the basis of distension and increased amplitude of pulsation of the cranial arteries, it is somewhat surprising that diseases associated with dancing of arteries such as aortic incompetence and severe thyrotoxicosis are not marked by headache more constantly than they are. It is also difficult to follow how the mechanism of vasodilatation causes pain in the head or headache. Pain related to blood vessels usually arises from vasoconstriction. Vasodilatation of a limb is not associated with pain. Why then is pain caused by vasodilatation of cranial arteries?

Artificial histamine headache.—The headache produced experimentally by injection of 0·1 mgm. of histamine phosphate has been found to be due to dilatation, stretching and increased amplitude of larger basal arteries. Branches of the internal carotid artery and the vertebral and basilar arteries are chiefly involved and determine the quality and intensity of headache. Investigations have shown that anything that tends to increase the stretch or diminish the support of the vascular walls tends to increase the headache and *vice versa*.

Pyrexial headache.—Pyrexial headaches probably have a similar origin and they have much the same characteristics as histamine headache.

Toxic headache.—In cases of toxic headache, the offending materials are transported by circulation from its source of origin to pain-sensitive structures of the head and headache is produced either by vascular dilatation or direct irritation of the pain-sensitive blood vessels or meninges. Chief toxic headaches are—

(1) toxæmias such as uræmia, eclampsia, cholæmia etc.
(2) pus and other products of infectious processes formed anywhere in the body when absorbed may cause toxic headache.
(3) over-indulgence in alcohol and excessive tobacco smoking.
(4) drugs and various chemicals such as general anæsthetics, narcotics, iron, digitalis, strychnine, heavy metals like arsenic, lead and mercury, gasoline, carbon monoxide etc.

The term auto-intoxication is a vague one. But it is possible that incompletely digested toxic materials are absorbed from the gastro-intestinal tract in certain run-down and chronically tired individuals often suffering from digestive disturbances and this may result in the development of headache.

Histamine headache or Cephalgia which resembles in some cases headache produced by subcutaneous or intravenous injections of histamine (not to be confused with similarly induced migraine) is being identified with increased frequency in recent years, though its existence is still doubted by many neurologists. Evidences are there to show that vasodilatation of the cerebral vessels particularly branches of internal carotid artery is responsible for this condition. It can be produced by injection of histamine and is abolished at the height of the attack by raising the intrathecal pressure, extramural support being thus given to the cerebral arteries.

Migraine.—In migraine, it is the extracranial (particularly the temporal) and the dural arteries which are chiefly concerned in the causation of headache. Direct observation of the temporal artery during an attack of migraine may show it to be dilated and pulsating strongly. Pressure on the carotid artery or on the temporal artery during the attack may relieve the pain. There is no evidence that the cerebral arteries are involved in this process. Ergotamine tartrate which influences the extracranial and dural arteries relieves dramatically the headache of migraine in the vast majority of cases. It has got no action on the cerebral vessels. The pain in migraine can be abolished by section or injection of the trigeminal nerve and in many cases only of the supra-orbital

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and/or auriculo-temporal nerves. The pain is relieved by interruption of sympathetic nerve supply to the arteries of the same side of the head. Such measures, however, do not prevent the occurrence of sensory phenomena which may precede or accompany the onset of headache since these seem to have their basis in disturbance of cerebral arteries. The focal distribution and unilateral character of migrainous headache have never been satisfactorily explained.

The headache at the third stage of migraine is, however, more generalised and often bilateral. The scalp over the affected area is tense and tender. This headache is supposed to be due to stretching of the scalp with œdema of the vessel walls and pressure on the pain-carrying nerves. As might be expected, when this stage is reached vaso-constrictors have no effect.

Hypertensive headache.—Hypertensive headache resembles migraine in respect of mechanism since it is the stretching, particularly of the extracranial arteries which is at the root of the headache experienced by hypertensive subjects. Such headaches however do not parallel the rise in blood pressure. Cases of very high blood pressure are also met where the patient does not complain of any headache at all. It seems likely that it is the emotional hypertensives who are more liable to headaches, *i.e.*, those who were apt to suffer from headache prior to development of hypertension.

Tension headache is again due to vasodilatation. Nervous tension causes early vasoconstriction which when long-continued gives way resulting in vasodilatation and headache. They respond to vasoconstrictors.

Other headaches of vascular origin—

(a) *Raised intracranial venous pressure* causes headache. Such raised pressures may be due to intracranial causes such as thrombosis of venous sinus or due to extracranial causes such as chronic emphysema, large goitres, and intrathoracic tumours.

(b) *Cerebral arteriosclerosis*—Headache is probably the result of productive and proliferative changes in the vessel walls and adjacent meninges causing unusual tension of the sensory end-organs in the vessel walls.

(c) *Arteritis of superficial temporal arteries* may cause very severe pain over the temporal regions.

(d) *Hypertensive encephalopathy*—Headache of increasing severity is an important symptom of hypertensive encephalopathy, the pathological basis of which is probably constriction of cerebral arterioles and capillaries ultimately resulting in œdema of the brain.

Variations in intracranial tensions.—In cases of increased intracranial pressure, the degree of headache has no relationship with the height of pressure. Very high intracranial pressures may also be artificially induced without causing headache. Headache commonly associated with intracranial tumours is often not due to increased intracranial tension but it is supposed to originate mainly from stretching or tension of the neighbouring pain-sensitive structures. A rapid change in the intracranial pressure, however, often provokes headache of considerable severity. Persistent headache may occur on insolation when the spinal fluid pressure goes high without any alteration in the character of the fluid. In hypertensive hydrocephalus where the spinal fluid pressure is increased without any alteration in the constituents of the fluid, the headache is found to fluctuate in accordance with the cerebrospinal fluid tension and is relieved following reduction of pressure by lumbar puncture. Stretching of the dura by accumulation of excess fluid may be responsible for the causation of headache.

A drop in the intracranial pressure is commonly associated with headache, as is usually found after lumbar puncture when there is a leakage through a large opening of the arachnoid and dura after withdrawal of the needle. The post-lumbar-puncture headache is supposed to be due to traction on the meninges rather than any type of medullary herniation through the foramen magnum. 'Low Pressure' headache sometimes occurs in persons suffering from anæmia and low blood pressure. It may also be induced in some individuals by administration of saline purgatives.

Space-occupying lesions within the cranium.—The headache commonly associated with space-occupying lesions is supposed not to be due to increased intracranial pressure for many instances of extremely high intracranial pressures are found which are unaccompanied by headache. Certain intracranial tumours of very slow growth may occupy a large portion of the intracranial space without producing a material amount of headache. The rapidity of development of the tumour not allowing for intracranial adjustment and escape of fluid from the intracranial space and the presence or absence of rapidly developing tension upon the dura and the great vessels and sinuses are more important factors in the development of headache. The mechanism of headache is still a matter of controversy. It may be due to stretching of dural membrane or due to abnormal tension in the walls of the intracranial blood vessels. The headache is present in some cases of tumour when there is no rise in intracranial tension and it may be produced or increased by lumbar puncture which lowers the tension. Such headaches are usually made worse by coughing, sneezing or straining which tend to increase the intracranial tension and to accentuate the existing strain on the neighbouring structures. Jugular vein compression often aggravates the headache owing to

increase in the mechanical factors which form the basis of these headaches. Compression of the carotid artery, on the other hand, tends to relieve it.

Pain due to local pressure of the growth is predominantly unilateral on the side of the tumour. As the intracranial pressure rises, the headache often tends to be diffuse. The complicating hydrocephalus may lead to paroxysms of severe diffuse pain radiating down the neck. Pressure on the trigeminal nerve causes unilateral pain most commonly along the distribution of its first division and associated with hypo- or hyperalgesia of the same area.

Post-lumbar-puncture headache is supposed to be due to continued leakage of spinal fluid through the lumbar puncture hole in the meninges into the surrounding tissues. This allows the brain to sag thus bringing about a stretching of the veins connecting the cortical surface with the sinuses and of the meningeal attachments between the pia-arachnoid and the dura particularly along the margins of the superior longitudinal sinus and these regions have been found to be highly sensitive to pain on traction. Increasing the pressure of the spinal fluid or lowering the patient's head will relieve the traction and stop the headache. Sitting or standing increases the traction and hence intensifies the headache.

Meningeal affections.—The headache in meningitis is due to stretching of the dural arteries, irritation of the pain receptors by chemical products of local inflammation and reflex stiffness of the neck muscles. Since the same symptom though less severe is present in meningism where there is only a rapid increase in the amount of spinal fluid and since headache is promptly relieved following reduction of spinal fluid pressure by lumbar puncture it may be that pain is due at least in part to the stretching of the dura mater by accumulation of excess fluid. Headache may be caused by direct irritation of the nerve-endings in the meninges by infective, suppurative, hæmorrhagic, toxic or abnormal metabolic products in the cerebrospinal fluid. Headache associated with leakage of blood into the subarachnoid space is due to irritation of the meninges by the extravasated blood. These headaches are associated with hyperalgesia of the scalp and other manifestations of meningeal irritation.

Diseases of bones of the cranium.—The pain and headache associated with osteomyelitis and other pathological conditions of the skull are apparently produced by involvement of the contiguous pericranium and dura mater.

Headache associated with extracranial causes at the head level.—Continued abnormal stimulations arising in structures of the head such as tissues and muscles of the eyes, impacted wisdom tooth, inflamed and indurated muscles of the scalp and neck, apparently by reason of their contiguity or particular relation to the head are capable of giving rise to sensations which are appreciated as headache. Similar conditions of over-use of muscles in a finger would never cause headache whereas an analogous stimulation of the intrinsic or extrinsic muscles of the eye gives rise to headache. There seems to be some inherent relation between painful conditions of the structures of the head and headache itself and many extracranial painful conditions may under certain conditions acquire a further elaboration of the pain and become a headache.

The important extracranial causes of headache at the head level are—

(1) Eye affections—Chief of which are irritation of the retina, diseases of the coats of the eye (including glaucoma) and eye strains caused by muscle imbalance, necessity for excessive accommodation etc.

(2) Affections of nose and nasal sinuses.—The inflammation of frontal, sphenoidal or ethmoidal sinus is a common cause of headache. Whether maxillary sinus disease causes headache or not is still a matter of doubt. So often a patient is seen whose maxillary antrum is full of pus but never complaining of any headache at all. Toxic headache may, however, be caused by absorption of enclosed pus from the antrum. Nasal obstruction apart from infection may be the cause of persistent frontal headache. Vacuum frontal headache, at one time a common explanation of headache arising from frontal sinus is not frequently met with in recent years.

(3) Diseases of ear, mouth, throat and teeth such as otitis, tonsillitis, root infections of the teeth, unerupted molar tooth etc.

(4) Diseases of the scalp and neck—particularly myofibrositis of the scalp and neck muscles and cervical spondylitis. Some cases of post-traumatic headache may be due to injury of the scalp.

(5) Neuritis and neuralgias in connection with supra-orbital, auriculo-temporal, posterior auricular and great occipital nerves.

Referred pain in the head from distant organs.—In certain visceral diseases, pain may be referred to the head from remote organs in the thorax, abdomen and pelvis. The explanation of this reference of pain is that the trigeminal nerve is the somatic sensory nerve corresponding to the vagus by which so many viscera are innervated.

Post-traumatic headache—Post-traumatic headaches may be caused by the development of adhesions between the various layers of the meninges or between the meninges and the cerebral substance itself. Adhesions in the subarachnoid space between the dura and the arachnoid is, however, the most potent cause of headache. The adhesion seems to develop from

organisation of a thin layer of blood extravasated into the subdural space. Whenever there is pull upon the adhesions, tension is transmitted to the region of entrance of the veins into the sinuses which are extremely sensitive and headache ensues. Anything which tends to pull the adhesion tends to cause or aggravate the headache.

Post-traumatic headaches may also be due to involvement of the nerves of the scalp in the scar tissue after laceration or contusion of the scalp. This, however, causes a superficial pain rather than true headache and this is associated with focal tenderness and sensory disturbance. A much rarer condition is post-traumatic atrophy of the brain which is responsible for the development of very serious headaches. The headaches associated with chronic subdural hæmatoma are of the brain tumour type to which reference has already been made.

Mackenzie attributes the usual post-traumatic sequelæ of headache and dizziness to a loss of vascular tone produced by protracted maintenance of recumbent position. He advocates assumption of semi-erect and erect postures and quiet walking in the ward as early as possible after the head injury unless there are evidences of actual brain damage and this regime according to him minimises the post-traumatic sequelæ.

Whether and how far the post-traumatic headaches are organic or psychoneurotic in origin is a matter of controversy. It is generally supposed that most of them have a basis in organic disturbance. In many cases, the anxiety and depression soon complicate the picture and these often aggravate the headache. Factors which increase attention or startle the patient such as concentration, prolonged reading, continued effort of any kind, loud sharp noises, etc., usually aggravate the headache. It is often extremely difficult to determine when post-traumatic headaches cease to be due to organic causes and are carried on by psychological influences and there is usually a wide margin of overlap between organically determined and psychologically determined post-traumatic headaches.

Psychological headache.—The neuroses form about one-third of all cases of headache appealing for medical aid and deserve special mention. In many cases the head symptoms in the neurotic follow a regular course. At first under the weight of adaptive strain headache is experienced. Then comes what is known as the psycholeptic attack. This consists of a sudden snap or bang apparently inside the head as if something under tension like a piece of elastic has given way. This causes extreme fear and distress which lead to typical head symptoms of psychiatry, not real headaches but odd head sensations, not so much painful but chiefly troublesome on account of their oddness and the fear of mind and brain disorder which they carry. They respond better to sedatives than to analgesics.

The mechanism of psychological headache is not still determined. In many cases, it is undoubtedly a conversion hysteria symptom but in others it seems to have some of the characteristics of vascular headache. The cerebral circulation is susceptible to nervous influences and it is quite possible that the cerebral circulation is so much disturbed in some psychotic individuals that they suffer from headache. A source of anxiety headache is supposed to be persistent contraction of the frontalis muscle. Wolff and others believe that tension in the muscles of the neck long continued as the result of emotion is the means by which headache is produced in many psychotic individuals. As might be expected, aspirin and other analgesics have practically no effect in relieving the psychological headache.

Physiology of Headache

Briefly speaking, headaches are produced principally in the following ways—

(1) Dilatation and distension of the arteries inside and outside the skull—commonly branches of internal and external carotid artery.
(2) Traction on vessels or meninges within the skull.
(3) Oedema and spasm of the muscles of scalp and neck.
(4) Direct irritation of the pain-carrying nerves and structures by inflammation or pressure (including the referred pain from distant viscera).

Distribution of pain in the head and its path to consciousness—It has been found that stimulation of a pain-sensitive structure above the tentorium produces pain in front of the inter-auricular line being carried by the sensory branches of the trigeminal nerve. Similarly, pain produced from structures below the tentorium is felt behind the inter-auricular line being conveyed by sensory branches of 9th and 10th cranial nerves and upper three cervical nerves. The role of sympathetic nervous system in the conduction of pain impulses is still incompletely understood.

Classification

On the basis of mechanism or physiological mode of production, headaches may be classified as follows:—

(1) *Vascular headache*—Resulting primarily from dilatation of blood vessels at the head level. Chief vascular headaches are—

(*a*) migraine.
(*b*) artificial histamine headache and histamine headache or cephalgia.
(*c*) tension headache.
(*d*) hypertensive headache.
(*e*) pyrexial headache.
(*f*) some toxic headaches.

(2) *Psychological headache.*—The mechanism of this is not properly understood. These are not real headaches but some odd head sensations which have no organic basis and do not result from disturbance of vascular function.

(3) *Intracranial headache*—Resulting mainly from traction or stretching of the meninges. In this group may be included the headaches which seem to originate from direct irritation of the nerve-endings in the meninges. Chiefly the following headaches belong to this category—

(*a*) drainage headache (after lumbar puncture).
(*b*) some headaches associated with space-occupying lesions within the cranium.
(*c*) some late post-traumatic headaches.
(*d*) headaches associated with anæmia and low blood pressure may be of this nature.
(*e*) some headaches associated with increased intracranial pressure such as those of meningism or hypertensive hydrocephalus may be due to stretching of meninges by accumulation of excess fluid.
(*f*) Headache in subarachnoid hæmorrhage.
(*g*) Some toxic headaches may be due to irritation of nerve-endings in the meninges by irritant materials in the cerebrospinal fluid.
(*h*) Some affections of the calvarium may cause headache by irritation of the meninges.

(4) *Extracranial headaches*—Resulting from irritation of the sensory cranial and cervical nerves outside the skull. This group includes the headaches which are referred from distant organs in the thorax and abdomen. The irritation chiefly arises from the following organs and areas:—

(*a*) nose and nasal sinuses.
(*b*) eye.
(*c*) ear, teeth, mouth and throat.
(*d*) scalp (including the periosteum) and neck.
(*e*) viscera in the thorax, abdomen and pelvis.

(5) *Mixed type*—Where more than one mechanism mentioned above are concerned in the causation of headache. In meningitis, there is stretching of the dural arteries and irritation of nerve endings in the meninges by products of local inflammation, as also reflex stiffness of the neck muscles. In post-traumatic headaches there may be injury of the nerves of the scalp as well as traction on adhesions of some intracranial pain-sensitive structures and quite often a psychological element it added to these causes.

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SOME DON'TS IN CARDIAC PAIN

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The term 'cardiac pain' is employed here only in a clinical sense—*i.e.*, as is described to us by the patient as 'heart pain.' Hence pains resulting functionally have had to be included with those hitherto considered as due to organic causes. Other forms of cardiac distress such as breathlessness, restlessness, weakness under the term pain have been scrupulously avoided.

When all is said and done, pain is not a feature of all diseases of the heart. For example, serious and even fatal forms of acute myocarditis can exist with little or no pain. On the other hand, there exist some important groups of heart pains that are extremely severe and so characteristic and so different from pains elsewhere in the body that their recognition should not be very difficult if a few fundamentals are borne in mind. These fundamentals have been formulated as "don'ts."

(1) In the absence of rheumatic infection (rarely other infections), *don't* be hasty to diagnose an organic cause for the heart pain in the first half of life. Congenital diseases of the heart may exist and go on without any pain unless complications supervene. Even in the rheumatic group, pain is present only in a certain percentage of cases in the early pyrexial stage—constantly with the onset of pericarditis, frequently in aortic insufficiency wherein the pain takes an anginal character, and not so frequently in mitral stenosis and then also only when valve cusps are considerably affected.

(2) *Don't* ignore functional causes of cardiac pain in this age group. Heart pains of functional origin are by no means uncommon. As early as the period of second dentition pains may be complained of and go on to school-age and later. Family history and set-up are important factors in the causation. Other factors include parental rigour and punishment, parental indulgence, presence of other patients in the house, the family talk, fear of examinations etc. This trouble can occur not merely in a spoilt child, but also in an impressionable child of neurotic diathesis.

Other heart pains which come on in adolescents and youths are of a mixed category of functional diseases, known variously as *effort syndrome, soldier's heart, neurocirculatory asthenia, cardiac neurosis* etc. The exact site of pain is indefinite—often more apical, supra-, infra-, and para-mammary rather than in the central zone of the chest; exaggerated epithets are employed in describing the pain. But a few acquire a smattering knowledge or even a very fair knowledge on the subject of anginal type of pain. The medical profession appears to be well represented in the latter category. Shock, a neurotic diathesis, illness, effort

in adverse circumstances—all play a part in induction of this type of pain.

(3) *Don't* deny them a thorough examination before arriving at a diagnosis and *don't* fail to note the eight cardinal points of Sir Thomas Lewis, same as you do for any organic heart disease, *viz.*,—

1. Exercise tolerance. 2. Evidence of congestion with or without failure. 3. Enlargement of the heart size. 4. Rhythm. 5. The state of the valves. 6. Evidence of infection. 7. State of the vessels. 8. Etiology.

A stethoscope and blood pressure apparatus should do, but some are so extravagant in their demands that x-rays and electrocardiogram should be called into aid when available, if only to impress the patient or to avoid possible errors in diagnosis.

(4) *Don't* fail to be emphatic in giving a good prognosis though it is a well-known fact that prognosis is uncertain for the symptoms, yet good for life.

(5) *Don't* fail to search the thyroid as the cause of pain especially when the pain is accompanied by tachycardia, increased B.M.R., and pulse irregularities. Masked hyperthyroidism is by no means rare with thyrotoxic hearts. The pain is often anginoid, and the prognosis is very favourable with suitable and timely treatment.

We now come to two important clinical conditions —angina pectoris and coronory occlusion—that produce true heart pain having the same basis of origin, *viz.*, coronory insufficiency and myocardial anoxia. We need not go far into the many views regarding the theories of causation. Suffice it to say that as clinical entities they deserve careful study.

(6) *Don't* expect much objective findings in angina; careful clinical history is more important. Effort or exercise, walking up a gradient or stairs, exposure, emotion, excess of food or drink etc., are precipitating factors for the attack. On the other hand, rest affords immediate relief; paroxysmal occurrence of pain with comparative freedom between attacks is the rule. Duration of pain is often less than five minutes. Pain lasting over fifteen minutes is most probably not Heberden's angina. It is more likely to be one of coronory occlusion. Pain frequently radiates to left shoulder, arm and wrist; But both arms and back may be involved if severe. Pain usually is one of constriction, compression or strangling nature, but not throbbing, dull or pricking. There is welcome relief with nitrites. Women suffer far less unless suffering from thyrotoxic heart, high blood pressure and diabetes. In association with anginal pains, emotional upsets ranging from anxiety and fear to 'angor animi' are usual. Co-existing cardiac disease such as hypertension, and arteriosclerosis would be helpful points in diagnosis.

A serious variety of angina may occur with little or no effort thus meriting the name *status anginosus*. As the patient is very ill and other cardiac signs and symptoms are evident, it needs but a brief reference. The prognosis is grave.

The subject of differential diagnosis of angina is vast involving nearly every contiguous structure of the heart and chest. To recall a few:—Pre-herpetic pains of the chest more likely to be mistaken for coronary thrombosis, fibrositis, myositis, neuritis and neuralgia, arthritis, cervical rib, spondylitis, bursitis, pericarditis, pleurisy (both diaphragmatic and mediastinal), cancer of the lung, indigestion (particularly flatulent type), aortic aneurism, old rib and costal cartilage injuries, gall bladder diseases, aneurysmal dilatations of the aorta, syphilitic aortitis, spinal troubles, radiculitis, peptic ulcer, severe anæmias of all types, tobacco angina, hyperadrenalinism, œsophageal spasm and diverse group of neurosis already referred to. X-rays and electrocardiogram, blood counts and serological examinations may all be needed. An early diagnosis of the nature of anginal pain, however brief it may be in duration but related to effort, lacking in radiation with unequivocal cardiac signs, will help the treatment.

(7) *Don't* fail to impress on the anginal patients the possibility of a hopeful outlook if they lead a sensible life by practising moderation. *Don't* ever mention the word sudden death to the patient, though this fatality occurs in ten to fifteen per cent of the sufferers. Many patients of uncomplicated angina may live even for ten years. Good advice is more important than any medicine if only to disabuse the patient's belief that medicines can make good for dietetic excesses and regulated life. Nevertheless, nitrites, xanthine group of drugs and sedatives are usually administered. Paravertebral alcoholic injections are very useful in severe pains and hence the technique deserves to be mastered.

(8) Finally, *don't* let your anginal patient ever be without trinitrin tablets handy.

One rare form of angina needs mentioning only because it occurs in the younger group of people. Described first by Nothnagel, it is now known to be purely a vasomotor phenomenon and has nothing to do with the heart and has a good prognosis for life.

Acute coronary thrombosis, acute coronary occlusion and cardiac infarction—these conditions form a distinct clinical picture of true cardiac pain. It is even more important than angina pectoris as a clinical study. In many ways, the pain is similar to that of angina and yet differing in certain fundamentals. Occurring independently of effort, its severity and longer duration, failure to respond to nitrites and the

presence of severer shock are the distinguishing features.

The three terms are different from one another. Often a distinction is made between coronary thrombosis and coronary occlusion. Coronary thrombosis is thrombosis of a major coronary artery whereas coronary occlusion is due to either a thrombosis, embolism or internal hæmorrhage, and cardiac infarction may occur as a result of the above two conditions or independently as a result of sudden decrease in the volume of oxygen in the coronary blood (as in circulatory or hæmorrhagic disturbances). Not all cases of coronary occlusion go on to cardiac infarction. Hence the characteristic electrocardiographic changes do not take place in occlusions without infarction.

The importance of the subject lies in the fact that as cause of death, heart diseases come very high and cardiac infarction and allied causes account for a large percentage of cardiac deaths.

TREATMENT SERIES

MEDICAL MANAGEMENT OF CHRONIC RHEUMATIC DISORDERS

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One of the bugbears of medical practice is chronic rheumatism. Disappointments, frustrations and disreputations have fallen to the lot of the physician who has handled a number of such cases. From one medicine to another, from dietetic regime to regime and from this physician to the other, has been the pitiful movement of the chronic rheumatic patients, seeking restlessly for a cure or relief. There is hardly any other disease where amateur or expert psychotherapy, various forms of physical therapy, climatotherapy, diet regimes, talismans, herbal curiosities, and even the exorcist's powers have been tried so often with so little impressive overall results.

From the therapeutic point of view chronic rheumatic disorders may be classified as—

1. Articular rheumatism,
2. Non-articular rheumatism.

Articular rheumatism may be of two types—

1. Rheumatoid arthritis (Eli Type I, Fisher type II or synovial form).
2. Osteo-arthritis (Eli type II, Fisher type I or chondro-osseous form).

As proposed by the Committee of the British Medical Association for rheumatic disease (1933), gouty arthritis, pyogenic arthritis, arthritis of specific infections, rheumatic arthritis, toxic-metabolic, hæmophilic and allergic arthritis and various neuro-arthropathies are excluded from the category of chronic rheumatic disorders and, therefore, will not be discussed in the present paper.

Non articular rheumatism, according to Freyberg (1951), may be classified as—

Fibrositis—generalised.
Fibrositis—localised.
Painful stiff-neck.
Lumbago.
Bursitis.
Tenosynovitis (including Dequervain's disease).
Tendonitis—Trigger finger.
Tendon attachment syndrome.
Fasciitis (including Dupuytren's contracture).
Peri-arthritis (para-arthritis) (In shoulder—frozen shoulder).
Panniculitis.
Herniated subcutaneous fat.
Reflex neuromuscular dystrophy.
" Shoulder-hand " syndrome.
" Psychogenic rheumatism." (Rheumatic manifestations of psychoneurosis).

Treatment of Acute Exacerbations of Chronic Rheumatism (Articular or Non-articular)

During acute attacks, the object is to alleviate the pain which is often very severe, and to relieve the strain from the affected region by securing proper immobilization of the part. Relief of pain may ordinarily be provided by analgesic sedatives like codeine, acetyl salicylas, phenacetin and pyramidon, the latter being used rather cautiously in view of agranulocytosis, which may occasionally result from prolonged use. Rarely in severe cases omnopon, nepenthe, pethidine and even morphine may be necessary for relieving the acute pain. The writer finds I.M. injections of a pyrazolidine compound (irgapyrine) (5 c.c. every other day × 5 such) useful for lessening the pains.

Immobilization may be secured with proper splintage or less effectively with the help of pillows and sandbags suitably arranged on the bed. It is universally recognised that although sodium salicylate is extremely useful in rheumatic infection, it is much less beneficial during exacerbations of chronic rheumatoid arthritis or of non-articular rheumatism. Colchicine used in gout has no use in rheumatoid arthritis but may occasionally be beneficial in cases of fibrositis with hyperuricæmia.

Penicillin and sulphonamides which have proved so useful in pyogenic arthritis are hardly of any benefit in the forms of rheumatism under discussion.

Local injections of procaine solution (2 to 10 c.c. of a 2 per cent solution) are temporarily useful in allaying the pains of non-articular rheumatism.

Infra-red ray therapy (for 10 minutes from a distance of 10 inches) over the affected part twice a

day makes the patient grateful in most cases of non-articular rheumatism and in some cases of articular rheumatism.

Short-wave diathermy, where available, is the best means of delivering heat to joint tissues in cases of articular rheumatism. Early massage of the local tissues along with heat therapy may be beneficial in non-articular rheumatism after local infiltration of novocaine. Cortisone and ACTH, both used singly, are extremely helpful in controlling the symptoms in acute exacerbations, allaying pain and stiffness, decreasing the local muscle spasm and increasing the range of movements of the affected region, chiefly in articular rheumatism but also in non-articular rheumatism. Being costly, they are much less likely to be used in the latter group of cases.

The present position of cortisone and ACTH therapy in rheumatic disorders is discussed separately.

Treatment of Chronic Rheumatism

Articular and non-articular rheumatism require separate discussion.

Rheumatoid Arthritis:

Principles of treatment are—

General—1. To protect the tissues from the effects of factors causing collagen disease by endocrinal therapy *viz.*, cortisone and ACTH.

2. To maintain a high level of general health and to guard against factors aggravating or precipitating rheumatism—cold, damp, fatigue, trauma, infection including focal sepsis, psychical disturbances, postural defects and muscle imbalance.

3. To prevent local muscular atrophy by movement, massage and diathermy.

4. To prevent flexion contractures—by movement, massage and diathermy and by proper splintage in the best functional position.

5. Relief of aching and pains by Cortisone and ACTH. { codeine / iodine / salicylates I.V.

6. Correction of deformity and contractures ... { by

7. Restoration of functions of joints ... { orthopedic measures including surgery.

General measures—In every case, an attempt should be made to improve the general health and maintain it at a high level by adequate rest, a balanced nutrition, fresh air, iron and vitamins. Mental tranquillity should be quickly restored and a will and confidence to get well have to be developed. Fatigue should be avoided in every case. Bowels should be kept regularly moving preferably with a regulated and balanced diet and inculcation of good habits. Laxatives should be sparingly used. It should be remembered that there is no known " specific anti-rheumatic " diet. Plenty of green vegetables and fruits should be included in the dietary which should contain adequate quantities of proteins and which should provide adequate calories. Supplementary multiple vitamins should be given to the patient with chronic rheumatic diseases. Vitamin C is specially indicated as it has possibly some action in retarding the fibrinoid degeneration seen in the collagen diseases. The writer uses 500 mgm. to 1,000 mgm. of vitamin C daily for the treatment of the rheumatic patients, either orally during the chronic stage or intravenously during the acute episodes.

Blood transfusions and intravenous iron therapy may be necessary in the resistant anæmia of rheumatoid arthritis.

Graduated non-fatiguing exercises are beneficial to the rheumatoid patients and in cases of spondylitis deep breathing exercises should be encouraged, (for 5 minutes three or four times a day). This prevents fixation of the costovertebral joints and maintains an effective thoracic respiration.

Treatment of the septic focus—The old practice of sacrificing the teeth and tonsils and draining the nasal sinuses in chronic rheumatic disorders is dying out rapidly and justifiably as such eradication has not helped the patient to any appreciable extent. However, if there are any obvious septic foci, these should be removed, where possible, to improve the general health of the patient. In some cases autovaccines may be useful for controlling the severity and duration of activity of the septic foci and may be recommended along with other measures.

In many cases it should be remembered that focal sepsis is not the cause of lowered general health but is an ill effect of the same.

Drug therapy—Cortisone and ACTH.* In 1949, Hench and his associates described the remarkable effects of cortisone and ACTH in rheumatoid arthritis and of cortisone in rheumatic fever. Freyberg (1950), Boland (1950) and many others since then have confirmed these observations. It is very likely that these agents would be amply used in India in the foreseeable future and data would be available for analysis but it must be admitted that for most of the patients the cost at present is prohibitive. Also for many they would not prove useful—those unfortunate patients who have started their illness some years ago and are now in advanced stages of structural bone and joint defects.

What may be expected with Cortisone ACTH therapy?

A considerable diminution in stiffness and aching and then in tenderness and swelling of the joints is

* Cortisone-Cortone—Merck & Co. ACTH—Armour Lab.

likely to occur within a week or so. The erythrocyte sedimentation rate gradually decreases and hæmoglobin content increases.

The range of movements increases satisfactorily. Grip, hopping and standing on the toes may become possible with lessening of the local deformity. Even in ankylosing spondylitis, movements may increase. Subcutaneous nodules, if any, may melt away. Flexion deformities may be prevented if the drugs are given within 6 months of onset of the disease.

What is not to be expected from Cortisone and ACTH?

It has to be borne in mind that no reversal of gross bone and joint damage can occur under this therapy. Subluxations, contractures, ankylosis and calcification of ligaments are irreversible.

Doses: Cortisone—Injections of 100 mgm. twice a day, are given intramuscularly, for 3 days. Then 100 mgm. once a day only, for 3 weeks. Then 100 mgm. is given every other day and still later 50 mgm. thrice a week and this has to be maintained.

Intermittent courses have also been suggested. The maintenance therapy can also be effected by orally administered tablets (Freyberg and others, 1950). For children with Still's disease, the same doses are used as given for the adults.

ACTH may be given 60-100 mgm. daily (in four divided doses). The maintenance doses may be 25-40 mgm. every other day.

Cortisone vs. *A.C.T.H.*—Certain differences of activity of these two substances should be stated. Absorption and body distribution of ACTH is more rapid than that of cortisone. Therefore, ACTH is given 3-4 times a day (Armour & Co. has found a long-acting form of ACTH). ACTH being a protein may cause a foreign protein reaction while no such reactions are to be expected from Cortisone therapy. ACTH may contain traces of posterior pituitary hormones. The activity of the two agents differs, weight for weight. About 40 mgm. of ACTH is equivalent to 100 mgm. of Cortisone. Action of ACTH is manifest within 12 hours of administration. With Cortisone it usually takes 48 hours. ACTH is ineffective in the presence of Addison's disease where Cortisone should be used.

Salicylates in rheumatoid arthritis—It has always seemed curious that the response of rheumatic arthritis and rheumatoid arthritis to salicylate therapy differs so much. This has become all the more unintelligible in view of the recent work which suggests that salicylates exert their effects upon the suprarenals indirectly through the pituitary axis, leading to hypersecretion of adrenocorticosteroids (Cauwenberge, 1951). Roskan *et al.* (1951) found a significant decrease in the number of circulating eosinophils for more than 4 hours after an intake of 4-6 gm. of sodium salicylate. This is preceded by a significant increase of the urinary uric acid-creatinine ratio. Cortisone and ACTH have proved useful both for rheumatic infection as well as rheumatoid arthritis. If salicylates act through pituitary-adrenal axis, why is this difference in response when salicylates are administered in those two conditions?

The writer believes that there is a difference in absorption of orally administered salicylates in these two conditions, absorption being effectively adequate in rheumatic infection but completely inadequate in rheumatoid infection. This view at present is a speculative one and requires examination by laboratory studies. In the experience of the present writer intravenous administration of salicylates along with hexamine* has often yielded satisfactory clinical results where oral therapy of salicylates failed to impress. The writer is not convinced of the value of succinyl salicylates in these circumstances.

Gold therapy—In the absence of Cortisone and ACTH this may still be used with some temporary benefit particularly in cases not older than one year. Pains and stiffness, swelling and tenderness may lessen definitely and the raised sedimentation rate is lowered under this therapy. The writer has used weekly intramuscular injections of sodium aurothiomalate** (50 per cent aqueous solution). Starting with 10 mgm., the dose is gradually increased to 50 mgm. which is kept on for a number of weeks until a total 1,000 mgm. are given. Thereafter 50 mgm. can be given twice a month for a period of two or three years. One should be carefully vigilant for the toxic effects. Long term results of gold therapy are not impressive. Ragan (1951) does not find any appreciable result in the 5 year or more follow-up studies.

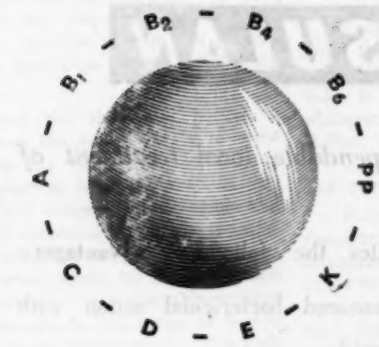
Iodine therapy in rheumatoid arthritis—This time-old remedy has been used by the writer in a number of cases and he recommends the use of the same in cases which cannot afford to procure Cortisone and ACTH. Injections are given I.V. diluted with glucose 12½ per cent 10 c.c. every other day, in gradually increasing doses: 1/4 c.c., 1/3 c.c., 1/2 c.c., 1 c.c. and 1 c.c. repeated, until total 8 or 10 injections are given. Calcium and Iodine† in 2 c.c. to 5 c.c. doses may be given intramuscularly or intravenously with less impressive results. The drug lessens the pains and stiffness, controls the fever, if any, and brings in a general sense of well-being. It is quite a cheap drug well within the means of the average patient.

Non-specific protein and fever therapy—In general practice, a number of preparations are used for this purpose—milk, milk with iodine, milk and sulphur, collosol sulphur, sterodin, omnadin etcetera. Intra-

* Cylotropine (Schering) or Pyelopurin (Cipla).

** Myocrisin (May and Baker).

† Iodine (P. D. & Co.) gr. 1/5 per c.c. (amp. of 1 c.c.). Calcium cum Iodine—Albert David 5 c.c. amp. and 2 c.c. amp. 2 c.c. amp.



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venous injections of T.A.B. are also being used by some. There is no doubt that at times non-specific protein therapy alleviates joint pains, stiffness, swelling and tenderness. In the absence of Cortisone/ACTH one or two of these preparations may be given a trial with some benefit.

Local measures—Non-surgical and surgical orthopædic measures. This is not within the scope of the present paper to deal in details these aspects of treatment which are readily available in any recent volume on orthopædics and in practice the orthopædic surgeon should be consulted in this respect. However, the following directions may be beneficial to the general physician managing the rheumatic patient entirely on his own responsibility:

It is desirable to give the rheumatic patient a firm bed with an overhead bar and side rails to assist the patient in changing his position. The foot is maintained in neutral position between inversion and eversion, the ankle in full range of dorsiflexion and the knee in full range of extension. The hip is maintained in the neutral position of rotation. The fingers are kept free but the wrist should be in dorsiflexion and the forearm in supination. It is desirable to have two shells for the elbow—one in full range of flexion and the other in full extension (Wells, 1951). The limb is taken off the splint daily once at least. The joints should be moved passively through their painless range at least once a day. Gradually such take-offs from the splintage are increased in number and duration and later the splints are discarded entirely (Wiles, 1949). Active movements are gradually superadded to the passive movements. If ultimate ankylosis is feared one should have it in the position of election. Graduated exercises of all muscle groups preferably under warm saline baths are definitely beneficial. Shortwave diathermy is the best means of delivering heat to the joint tissues and dilating the deeper vessels. Superficially applied heat may also be helpful in this respect but indirectly through a reflex vasodilatation. Immersion or contrast bath may also be similarly useful. Gentle massage of the muscles around the affected as well as non-affected joints delivered in the directions of venous flow is also beneficial.

Movements, massage and heat therapy are helpful in loosening up the muscles and joints and in opening up venous and lymphatic flow around a joint. When the patient's movements involve weight-bearing on the affected joints, crepe-elastic bandages, braces and corsets and weight-relieving calipers may be used, with advantage. Insertion of pads, wedge and lifts in the shoes may remedy postural defects (Wiles, *loc. cit.*). Manipulation under anæsthesia and done cautiously to avoid fractures may be undertaken to correct contractures and flexion deformities. Rehabilitation of the crippled patients should be the aim of all local therapy, and suitable work should be provided when he is fit for any.

Various surgical measures are applied with benefit in the advanced stages of the joint pathology. Skeletal traction for contractures and flexion deformity and synovectomy, capsulectomy, arthrodesis, arthroplasty, lengthening of contracted tendons, osteotomies, joint denervation and sympathectomy constitute a formidable list under the command of the orthopædic surgeon. As a physician, the writer hopes that in the future less and less of such surgery would be required for the arthritic patients adequately managed with medical measures.

Local deep X-ray therapy—The results of deep x-ray therapy to the whole spine and sacro-iliac joints in ankylosing spondylitis are very satisfactory. Its value is so great that it should be used in every case, whatever the stage of the disease (Dunlop, Davidson and McNee, 1949).

TREATMENT OF OSTEO-ARTHRITIS:

Relief of pain is secured by means discussed under rheumatoid arthritis. The local non-surgical measures advocated for rheumatoid arthritis are equally applicable to osteo-arthritis. Rest, avoidance of strains, correction of postural defects, application of local heat, light massage of muscles and graduated exercises, are as important points in the management of the osteo-arthritic as in the rheumatoid. Intra-articular injection of cortisone (50 mgm. in 2 c.c. may be helpful for the acute pains (Margolis, 1951).

In contrast to the rheumatoid patient, the osteoarthritic patient is usually well-nourished and obese. This obesity, if present, should demand a restriction of diet (800-1,200 calories) and supplementary administration of thyroid extract (gr. 1 b.d. or t.d.) or amphetamine preparations to depress the appetite.

Orthopædic appliances like braces for knees and the spine may be necessary for relieving weight-bearing.

The writer has no expriences of the intra-articular lactic acid (10 to 20 c.c. of 0·2 per cent solution in 2 per cent novocaine) therapy of Waugh (Dunlop, Davidson and McNee, 1949) but Wiles (1949) warns, in this regard, of the possibility of suppuration within the joint. Surgical measures like patellectomy, with or without synovectomy, in the knee cases, obturator neurectomy, arthrodesis or vitallium cup arthroplasty in the hip cases and spinal fusion in localised lumbar or lumbosacral osteo-arthritis are currently in use (Margolis, 1951). In localised osteoarthritis and chronic menopausal arthritis, deep x-ray therapy by a skilled radiologist, may be beneficial in relieving the symptoms (Dunlop, Davidson and McNee, 1949).

Of the drugs parenteral collosol sulphur therapy is mentioned to be of some use by Wagoner (1950).

TREATMENT OF NON-ARTICULAR RHEUMATISM:

In non-articular rheumatism, the remarks made under general management and local non-surgical measures for rheumatoid arthritis hold good. Of the drugs, intravenous salicylates, iodine or calcium *cum* iodine therapy and intramuscular non-specific protein therapy usually yield satisfactory results. It appears that Cortisone and ACTH are useful for non-articular rheumatism but for reasons stated before they are less likely to be used in such cases.

Vitamin E in 10 to 25 mgm. doses daily, has been used by some observers but in the writer's experiences results are not impressive enough to warrant further use.

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SPECIAL ARTICLE

CHRONIC FLUORINE INTOXICATION —A REVIEW*

PART I.

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POISONOUS EFFECTS

Fluorine and several of its compounds are poisonous to the animal and plant kingdom.[1] The effect may be local or systemic. *Exposure* of the skin for a fraction of a second to gaseous fluorine causes burns similar to those caused by an intensely hot flame.[2] Hydrofluoric acid has a delayed action[3, 4, 5, 6] but causes slow-healing painful burns. *Inhalation* of air containing 100 p.p.m. (parts per million) of fluorine proved toxic to experimental animals. Lower concentrations did not kill but were able to damage the respiratory system[7]. Inhalation of air containing hydrofluoric acid caused inflammation of the mucous membranes of the respiratory tract, œdema of the lungs and also degenerative changes in the testicles and subcutaneous hæmorrhages[7]. The deaths that occurred during a persistent fog in the Meuse Valley in 1930 are attributed by Roholm[6] to the presence of hydrofluoric acid in the atmosphere. Inhalation of air laden with the dust of fluorides causes acute pulmonary symptoms. *Ingestion* of fluorides and silicofluorides may cause acute or chronic symptoms according to the dose. The main symptoms of acute poisoning are severe irritation of the gastro-intestinal mucosa, paralysis of the central nervous system and musculature followed by death. Acute intoxication is caused either because of accidental swallowing or because of suicidal motives. Roholm[5] lists 112 cases of acute poisoning that occurred between 1873 and 1935 while further cases upto 1938 have been listed by Greenwood.[7] Mistaking an insecticide for milk-powder caused (in 1943) acute poisoning in 263 persons of whom 47 succumbed[8]. The use of baking powder containing fluorides caused gastric upsets[9] while apricot preserves contaminated with fluorides were responsible for widespread mass intoxication[10].

CHRONIC INTOXICATION

(i) *Extent in India*—While acute intoxication is rare, chronic intoxication of men and animals is fairly wide-spread and is generally traceable to the use of drinking water containing small (1·5 to 6·0 p.p.m.) amounts of fluorides. Endemic areas of fluorosis are met with practically in all parts of the world. In this country, chronic intoxication attributable to water-borne fluorides has been reported from parts of Madras[11], Travancore[12], Ceylon[13], Hyderabad (Deccan)[14], Mysore[15], United Provinces[16], Punjab[17, 18] and Baluchistan[18].

(ii) *Vectors—Foods; Factories*—In the case of human beings, the foodstuffs of plant or animal origin do not appear to play any significant role, the main cause of fluorine-intoxication being the drinking water. Industrial processes can account for chronic poisoning among those workers whose duties expose them to the dust of fluoride-containing materials[9] although contamination of food or water by the fumes or effluents may sometimes cause a hazard[19, 20, 20a].

(iii) *Vectors—Intoxication of cattle*—Drinking water containing fluorides may cause chronic intoxication in cattle, sheep and other domestic animals. In certain areas, the suspended matter in the water may play an important part.[21] The use of fluorine-containing

* This communication deals with the possibilities and symptoms of chronic intoxication by fluorine, reviews the present state of our knowledge on the mechanism of this intoxication and discusses the methods available for amelioration.

phosphates as mineral supplements may give rise to symptoms of chronic intoxication. Cattle may develop severe symptoms of chronic intoxication by grazing on pasture contaminated with volcanic ash or fumes from certain industrial processes such as the manufacture of superphosphate or of aluminium. Numerous such instances dating as far back as 1891 have been reported and are referred to in a study conducted under the auspices of the Medical Research Council of Great Britain in 1944 at an aluminium factory in Scotland[23]. Cases of chronic intoxication of cattle attributable to grazing on herbage contaminated with the fumes from burning of certain coals[20] (as in smelting of iron-ore) or clays[22] (as in brick-making) have been reported. In view of the fact that aluminium and fertiliser factories are springing up in India, it would appear desirable to keep a look-out against this possible hazard.

Symptoms of Fluorine Intoxication

A host of conditions (such as the irritative conditions of the gastro-intestinal and renal tracts, constipation, low blood pressure, mottling of the nails, fragility of the hair and alopecia, goitre-like changes and so forth) have been attributed to the ingestion of small amounts of fluorides[24, 25, 26]. However, the evidences in regard to these are not quite conclusive and the most characteristic symptoms of chronic fluorine-poisoning in human beings and domestic animals are the production of characteristic changes in the (1) teeth and (2) the skeletal system. Administration of fluorides or of mineral supplements (such as rock phosphate) containing fluorides to experimental animals have provoked symptoms of toxic intoxication such as a decreased rate of growth, loss of weight or even a reduction in the period of survival. But as far as can be gathered from the published records of surveys, such symptoms are not common among human beings in the endemic areas.

(1) *The Dental Changes* known as the "mottling of the tooth-enamel" were described as early as 1901 but it was not until 1931 that its association with the presence of fluorine in water supplies was first established almost simultaneously by three independent workers[27, 28, 29]. The condition is characterised by chalk-white transverse striations which first appear on the incisor teeth. The severity may vary from these mild chalk-white striations or a diffuse opacity affecting the entire tooth to a condition where the teeth show a characteristic yellow discolouration and pitting and chipping of the tooth enamel. *Animals* display—besides mottling—an abnormality in the size and shape of the teeth. The incisors become spatulate in sheep or tend to curve inwards (as in rats). The teeth do not oppose each other properly and render mastication difficult. The molars show an abnormal wear. Frequently some teeth are abnormally big while other teeth do not grow.

Surveys carried out in various countries showed that the severity of mottling bears a close relationship to the concentration of fluorides in the water supplies. On the evidence of the surveys among children, American workers laid down 0·85 p.p.m. fluorine as the permissible limit in a water supply intended for drinking purposes[30]. More recent evidence appears to have warranted the raising of this limit to 1·5 p.p.m. in America.

Fluorides and Dental Caries.—Dean discovered that the prevalence of dental caries among children drinking a water containing small amounts of fluorine was much less than that amongst children of similar age groups in areas where the water-supply contains none or less than 0·5 p.p.m. of fluorine. This discovery—which was also confirmed by numerous surveys in various parts of the world—has led to the suggestion that the *addition* of fluorides to the water-supply may offer a method of controlling the incidence of dental caries which is fairly high in America. Animal experiments have also confirmed the beneficial effect of fluorides in combating the incidence of artificially induced dental caries. It is suggested that by controlled addition of fluorides to water supplies (so as to raise the level of fluorine to 1·0 p.p.m.) the incidence of dental caries could be reduced by 30 per cent without exposing the children to the risk of mottled enamel of the teeth. It is also suggested that the ingestion of water containing such low levels of fluorine will have no other deleterious effects. As a result of these researches a number of communities in America are adding (or planning to add) fluorides to their water supplies. At several centres, the addition of fluorides to water supplies was started experimentally, one group of children receiving the fluorine-treated water while the other group continues to receive water free from fluorine. At one such centre—Newburgh, N.Y.—valuable pediatric studies have been inaugurated to study practically whether the ingestion of fluorides at these low levels has any harmful effect.

(2) *Skeletal Changes*—The other characteristic symptom of fluorosis is the formation of abnormal outgrowths (exostoses) on the long bones and the calcification of the intercostal ligaments thus giving rise to complaints of stiffness of the back and the neck. The first cases of such skeletal changes were noticed by Moller and Gudjonsson[31] in 1931 among cryolite workers. In his masterly study on this subject, Roholm[3] describes these changes in detail and presents evidence of his work as well as that of other investigators on the production of these characteristic symptoms in various experimental animals such as sheep, cattle, swine, guinea pigs, and rabbits.

Roholm distinguishes three phases of this process: In the *first phase*, changes were noticed only in the columna. The density of the bone was very slightly increased. The trabeculæ were rough, blurred and

gave deep x-ray shadows but the bone contours were sharp. In the *second stage*, the bone structure was blurred and the trabeculæ merged together. The columna showed incipient calcification of the ligaments while the medullary activity was moderately narrowed in the bones of the extremities. In the *third stage*, the bone contours are wooly and blurred and there is considerable calcification of the ligaments. Palpation shows the bone surface to be rugged. Hard indolent nodules up to the size of a pea are noticeable in the subcutaneous edges of the tibia and the ulna. The x-ray negative shows the bone as a diffuse, marble-white shadow in which the details cannot be distinguished. At this stage, motility of the columna—flexion, extension as well as rotation—is restricted. There may also be an increase in dorsal kyphosis and asymmetry of the shoulder region with atrophy of the exterior muscles of the back.

The bone of experimental animals receiving fluorine supplements assumes a chalky colour and lacks lustre. The thickness of the bone as well as the external diameter often show an increase while the surface is coated with a white irregular deposit.

BONE SYMPTOMS—FRAGILITY OF BONE: Several workers have reported that the long bones of animals (such as swine or cattle) receiving fluorine supplements suffer a change in their mechanical properties. Some have reported that the bones are rendered more fragile while others have noticed an increase in the breaking strength. No evidence of increased fracture experience have been noticed among men who showed mottling of their teeth enamel[3, 22]. Bell and de Weir[22] conducted a thorough study of the mechanical properties (such as the breaking and bending stresses, modulus of elasticity) of the long bones of sheep grazing on pastures contaminated by the fumes from an aluminium factory. These workers suggest that the alteration in the shape of the bone are such that the weakness caused by the effect of fluorides is compensated by an increase in the thickness and external diameter.

BONE CHANGES CAUSED BY WATER-BORNE FLUORIDES: Skeletal manifestations of fluorosis were not associated with the ingestion of the relatively small quantities of fluorine that could be ingested through water supplies until Short *et al*[33] first showed the existence of characteristic skeletal changes among the human populations in this country from an area of endemic fluorosis. Marshall-Day[34] found a high incidence of "stiff-back" among the older inhabitants in an area of endemic fluorosis in North India. Recently Khan and Wig[18] reported cases of skeletal involvement in two adults from an area of mottled enamel in the Punjab.

Following the publication of Shortt's findings, have come evidences of bone-involvement attributable to water-borne fluorides from China[35], Argentina[36], United States[37] and South Africa[38, 39]. Kemp *et al*[40] suggested in 1943 that cases of spondylosis deformans noticed by them among some children in the areas of Great Britain where mottling of the tooth-enamel is prevalent may be due to the progressive deterioration of the degenerative changes initiated by fluorine. Since x-ray studies have been employed only occasionally in the surveys of endemic water-borne fluorides, it is not improbable that the more frequent use of this diagnostic aid would reveal that the incidence of skeletal changes is greater than we now believe. Such a view is rendered more likely by the observation of Roholm[3] that these bone changes are insidious and do not give rise to subjective symptoms until the condition is so advanced as to impede natural movements.

CONDITIONS IN INDIA

The finding of skeletal changes in the areas of the Madras Presidency where the level of fluorine in the water supplies averages only 3 to 5 p.p.m. (maximum 10 p.p.m.) calls for an explanation. It would also appear that the severity of mottling of the tooth enamel is much greater in India than in the Western countries. Thus, while the mottling is described as "moderate" in parts of America where the fluorides are of the order of 2 to 2·5 p.p.m., such concentrations are associated in the Madras Presidency with severe symptoms such as chipping of the enamel. Again, mottling of the deciduous teeth of children has been noticed in these areas whereas such changes are associated with very much higher concentrations elsewhere.

It has been suggested that this increased severity of symptoms in this country is due to the poorer nutritional level of our nationals. Some evidence in favour of this view has been adduced by workers in this country[14, 17, 41] as also from those in other countries[42, 43]. It is also possible that climatic factors favour the consumption of larger quantities of water with consequent increase in the quantity of fluorine ingested. The role of the fluorine present in the cooking water is another point that merits investigation especially if it is remembered that foods, like rice, imbibe large amounts of water during cooking. Surveys which would help to assess the fluorine intake more correctly would be expected to clarify the issues but such an assessment is difficult because of the multiplicity of wells with varying concentrations of fluorine in their waters. It is likely, however, that the following up of the urinary excretion of fluorine among people in these endemic areas would offer a valuable clue.

The observation that the symptoms induced by fluorides are more severe in India suggest our attitude to the problem of fluorosis in two important directions, *viz.*, (1) our reaction to the idea of adding fluorides to water supplies as a method of inhibiting dental caries and (2) the orientation that we should give to the investigations on this subject in our country.

Addition of fluorides—Whether the increased severity of symptoms noticed in this country is due to

the lower nutritional status or to an increased intake of fluorine, it would appear to be prudent to adopt a conservative attitude to the question of following American experience either in raising the permissible limit in water supplies or in contemplating the addition of fluorides to water supplies until we know more about the subject as it affects the people under the conditions prevailing in our country. If fluorides should be used for the purpose of combating dental caries, the method of topical application of fluorides to the teeth would prove far more preferable.*

As far as can be judged from the few surveys in areas of endemic fluorosis in this country, it would appear that the incidence of " stiffness of the back " is appreciable. Although the changes in the shape of the long bones in sheep have been suggested as a compensatory mechanism, it cannot be denied that these peculiar rose-thorn-like growths recorded by Shortt *et al* in human beings and the calcification of the intercostal ligaments resulting in the impeding of natural movements can cause much discomfort. It would, therefore, appear desirable to shift the emphasis of investigations on unravelling the mechanism of formation of these " exostoses " and on exploring the possible methods of preventing their development.

Summary

Fluorine and its compounds can cause local as well as systemic symptoms of poisoning when inhaled or ingested by animals. While acute intoxication by fluorides is rare, chronic intoxication is widespread and is generally traceable to the use of drinking water containing small amounts of fluorides. In the case of cattle and other domestic animals the intoxication may be caused also by the use of phosphate supplements containing fluorides or by grazing on herbage contaminated by fumes of certain industries in which compounds of fluorine escape into the atmosphere.

The main symptoms of chronic intoxication due to fluorides are the characteristic (1) dental changes and (2) skeletal changes such as the formation of " exostoses " on the long bones and the calcification of the ligaments. Toxic symptoms (such as depression in the rate of growth or death) do not appear to be common.

The " inverse correlation " noticed between the presence of fluorine in water supplies and the prevalence of dental caries has led American workers to suggest the use of fluorides either as a topical application to the teeth or as a mass medication of the water-supply to combat this disease.

There is reason to believe that similar levels of fluorine in water supplies cause symptoms of greater severity in India as compared with some of the Western countries. Several causes—such as the lower nutritional level of Indians, the increased consumption of water caused by a hot climate—may account for this phenomenon. This increased susceptibility of Indians suggests the need for caution in accepting American experience in this field, while the fairly widespread prevalence of the bone symptoms suggests the need to shift the emphasis of investigations on fluorosis in this country to exploring the possible methods of unravelling the methods of preventing these troublesome changes. The rapid industrialisation of this country also suggests the need to keep a look-out for newer possibilities of fluorine-intoxication such as those caused by superphosphate and aluminium factories.

* This aspect has been discussed by the author in *The Indian Dental Review*, **18**: 289, 1949.

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CASE NOTES

FAECOLITHS AND FAECALOMAS WITH A CASE REPORT

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A fæcolith is a mass of inspissated fæces, dry and hard, formed as the result of bowel stasis which gives rise to accumulation of fæcal matter and a secondary process of desiccation. If the accumulation of fæces occurs rapidly and the process of drying is relatively slow, there results a soft, often pultaceous mass, to which the name fæcaloma, coproma or stercoroma has been given. On the other hand, if fæces collects slowly and the aqueous content is rapidly absorbed, a hard stone-like mass results to which the name fæcolith has been given. Fæcoliths or fæcalomas are usually of small size and occur commonly in the appendix where they may be responsible for acute appendicular obstruction. Moloney (1947), however, described an appendicular fæcolith of considerable size simulating gall-stone and producing a condition of paralytic ileus of the bowel. The large fæcoliths or fæcalomas are rarer and are usually found on the left half of the colon and most frequently in the rectum or in the sigmoid colon. On the right side *i.e.*, in the cæcum or the ascending colon, very few cases of large fæcoliths have been described. Walker (1948) described two unusual cases of massive fæcoliths in the cæcum which were radio-opaque. Blotner (1948) also described a case of cæcal fæcolith which caused local necrosis.

Fæcoliths have been responsible for mistaken diagnosis on various occasions and it must be unusual for a surgeon not to have come across a case sometime or other which he had feign put down as one of organic intestinal obstruction and which later on surprised him or his assistants by turning out to be only a mass of fæces: Weish and Brannon (1948) and Feldman (1942) described fæcoliths of large gut which simulated cancer. Rous (1945) gave details of a case of fæcolith in the transverse colon of a child which mimicked clinically as well as radiologically as a case of intussusception. Coleman (1944) described an appendiceal fæcolith which simulated ureteral calculus.

In women, fæcolith or fæcaloma formations are common during pregnancy and on various occasions they have complicated delivery and have necessitated cæsarean section (Baldi, 1941 and Ottow, 1940).

Etiology

The most obvious cause of fæcolith formation is chronic constipation and bowel stasis. There are, however, other factors which help their formation. Chronic inflammation of the large gut, such as localized diverticulitis, neoplasms especially malignant neoplasm of the large gut are often associated with inspissated fæces. It is significant that fæcolith formation in the large gut is most common in those areas which have long mesocolon, *viz.*, the sigmoid colon, the transverse colon and occasionally the cæcum. In Walker's (1948) two cases both had unusually mobile cæcum with long mesocolon. Rectal fæcoliths are not usually formed *in situ* but have travelled down from the sigmoid colon. The excessive mobility of the large bowel resulting from an unusually large mesocolon must interfere with the regular pattern of the peristaltic wave, thereby giving an opportunity to the fæces to accumulate and inspissate.

The following case is unique in my experience because the fæcolith in the sigmoid colon remained more or less stationary for well over 2 years and after its removal which included part of the bowel, a perfect result was obtained and the patient was able to divest himself of a colostomy which he was carrying for two years.

CASE REPORT

In September 1948, M.I., a Jewish male aged 50, complained of indigestion, occasional colicky pain in the abdomen and increasing constipation for about 2 months. He consulted a doctor who diagnosed subacute intestinal obstruction and a barium enema examination was performed. This revealed a persistent area of spasm and filling defect about the middle of the sigmoid loop in all the pictures (Fig. 1, *vide* Plate). The area was about 2½" in length and was somewhat tender. A diagnosis of a space-occupying lesion (?) carcinoma was made.

The patient was then operated on by a surgeon and a left iliac colostomy was performed. A piece of tissue from the

involved portion of the gut was sectioned and it showed evidences of chronic inflammatory tissue but no malignant changes. The patient thereupon carried on with his colostomy for about 2 years after which time he sought admission into the hospital and came under my care. He was very keen to have his colostomy closed as continence was unsatisfactory and he felt himself an outcast from society with it.

He was thin, emaciated, and the colostomy was working many times a day. A barium enema (Fig. 2, *vide* Plate) examination revealed that the area of filling defect in the sigmoid colon was present but possibly at a slightly lower level than in the original picture. Obstruction at this area was not absolute as irrigation through the distal colostomy loop caused fluid to flow per anum and *vice versa*. We were not very sure at this stage about being able to remove the obstruction in the bowel, reform the patency of the rectosigmoid loop and close the colostomy but impressed by the eagerness of the patient to be rid of his colostomy decided to explore.

On 8-6-50 the abdomen was opened through a left low paramedian incision. A hard mass of the size of a tennis ball was found at the lower end of the sigmoid loop. It was unusually smooth on the surface and one was struck by the total absence of surrounding adhesions. The bowel was clamped above and below by 2 pairs of clamps and the mass was removed. The continuity of the bowel was re-established by a single layer of everting interrupted seromuscular suture. The raw area in the pelvic cavity was peritonised and drainage established through a stab drain in front of the coccyx. The abdomen was closed in layers.

On cutting open the bowel wall of the mass removed, one got the shock of one's life to find that what we had removed was a mass of fæcolith. It was extremely hard, brittle and easily separable from the bowel wall.

Convalescence after the operation was uneventful and 4 weeks after this operation, the iliac colostomy was closed. Healing of wounds was good and the patient was discharged a month later with perfect continence re-established and he was passing fæces per anum. Follow-up of the patient one year later showed that the patient was in good health and that his bowel action was normal.

Discussion

Diagnosis in left-sided fæcoliths have often been difficult and often, as in this case, they were mistaken for inflammatory or neoplastic lesions. Clinically, the distinction may be impossible and so also radiologically. The latter method may be of some help for sometimes the fæcoliths may contain calcium and thereby produce a radio-opaque appearance. Also it may be possible to move the fæcolith slightly with barium enema administered with some pressure and after evacuation of the enema, it may remain coated with the dye (Cochrane, 1938). But the presence of an irregular area of filling defect, spasm and localized tenderness may all be common radiological signs both in neoplasms as well as in fæcoliths. The distinction may be difficult even after exploration. The features which characterize a fæcolith mass are its well-marked definition, roundness, smoothness and absence of surrounding adhesions or fixity. Continued presence of the fæcolith, however, for a time with associated obstruction of the bowel may produce secondary irritative and inflammatory changes in the wall and thereby lead to its irregularity, hardness, loss of definition and tenderness. Cronyn (1941) described a fæcolith in the right side of colon in an African which was deemed neoplastic and in which case diagnosis was not clear even on exploration. It was removed by enterectomy.

Summary

1. A case is reported where a massive fæcolith in the sigmoid colon eluded diagnosis for over 2 years and in which resection of the bowel with end-to-end anastomosis resulted in recovery of the normal bowel mechanism.

2. The literature is reviewed and the many interesting features of this condition are described.

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A CASE OF CAVERNOUS SINUS THROMBOPHLEBITIS

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Cavernous sinus thrombophlebitis had been a much dreaded condition as the cases almost invariably ended fatally. The discovery of penicillin had brought a cure for this condition, if treatment is commenced early. As there are very few cases reported in the literature, we feel it worthwhile reporting the following case which came under our observation and was cured with penicillin therapy.

CASE REPORT

G.C.M. age 35, years Hindu, male, clerk in S.D.O. Office was admitted on 10-7-1950 into the Eye Ward for the following complaints:

(1) continuous fever	since 5 days
(2) severe headache	,, 5 ,,
(3) proptosis, right eye	,, 3 ,,
(4) proptosis, left eye	,, 2 ,,

The history revealed that on 26-6-50 he noticed 2 boils over the face on the right side. On 2-7-50 he got fever accompanied by severe headache. The temperature was continuous ranging between 99°F and 101°F. There was no rigor. On 6-7-50 he noticed heaviness of the right eye and gradually on the same day he developed chemosis of conjunctiva and proptosis. In the local hospital he was treated for the fever. Next day a similar condition developed in the left eye and he was sent to this hospital.

On Examination—On the day of admission the following, were found: the patent was conscious; temperature 102°·8F, pulse 90 per minute. Both eyes—proptosed, lids œdematous; conjunctivæ congested, pupils of normal size and reacting to light, movements of the eyeball—restricted in all directions. Tension normal, visual acuity could not be exactly determined but was apparently good. Ophthalmoscopical findings, fundi normal except congestion of the retinal veins; discs were normal, no exudates or hæmorrhages.

Investigations—The blood examination showed: total W.B.C.—16,000, cmm.; differential count—poly. 86 per cent, lympho. 14 per cent; no M.P. was found. The urine was examined and nothing abnormal was detected.

The case was diagnosed as early cavernous sinus thrombophlebitis.

Treatment—Penicillin 1 lakh units started at 9 a.m. and repeated as 50,000 units every three-hourly. Glucose 50 per cent 50 c.c. I.V. B.D. On the 12th., the case was examined by the Professor of Medicine and the findings were: mental condition clear, no neck rigidity or Kernig's sign, no evidence of affection of the limbs (or paresis); tendon reflexes normal. Headache + + +, vomiting + + +, condition bilateral. Pulse 66 per minute, regular and of normal volume and tension. The above treatment (penicillin and glucose) was recommended and mag. sulph. 25 per cent solution 6 ozs. rectally by drip method was given. On the same day, 12-7-50, lumbar puncture was done—fluid was clear and not under pressure; pathological examination revealed no abnormalities. The blood pressure on that day was 110/70 mm. of Hg. (Day to day observation showed improvement of both the eyes and mental condition). Penicillin 30,000 units was given intrathecally. But subsequently penicillin was continued intramuscularly 3-hourly. Local treatment 4-hourly; hot fomentations to the eyes. Penicillin drops to the eyes. Black shades for protection of the eyes. Day to day observation showed improvement of both the eyes and mental condition.

The condition of the patient on 18-7-50: the temperature was normal, the mental condition clear, œdema and proptosis completely cleared up. On 19-7-50 morning, the temperature went up again to 102°F, headache and vomiting reappeared but the eye condition was normal. Penicillin was continued, Alkaline mixture, t.d.s.; cibalgin tablets when required and other symptomatic treatment were continued. The headache and vomiting got worse from 20-7-50 and the fever was fluctuating from 99°-103°F. On 27-7-50 blood was examined and the results showed—Total W.B.C.—68,000 cmm., neutrophil—61 per cent, lympho—32 per cent, mono—4 per cent, eosino—3 per cent, baso—nil, M.P.—not found.

Blood was examined also for microfilaria and it was negative. W.R. also negative. A skiagram of the skull was done on 28-7-50 and no bony changes were visualised. On 29-7-50 lumbar puncture was done for the second time and the result was: Fluid turbid and under pressure; Cell count—60 cells per cmm., D. C.—poly—30 per cent, lympho—70 per cent.

Chemical examination: Protein—100 mgm. per cent, globulin—Positive (+), chloride—760 mgm. per cent, sugar—20 mgm. per cent.

Culture—Meningococci grown (scanty colonies) Penicillin 40,000 units in 1 c.c. diluted in c.s. fluid was introduced through the L.P. route and the treatment then adopted was:

Penicillin 50,000 units I.M. every 3 hours, Sulphathiazole 2 tablets every 4 hours, Mist. Alkaline every 4 hours, Glucose 50 per cent 50 c.c. B.D., Civimin tablets 1 B.D., Nicotinic Acid tablets 1 B.D. On 31-7-50 the temperature came down to normal and on 2-8-50 headache and vomiting subsided completely. On the same day penicillin was stopped but sulphathiazole was continued. From 5-8-50 no medicine was given and the patient was given rice and milk.

The patient was discharged cured on 8-8-50. Visual acuity on discharge: B.E. 6/6. Ophthalmoscopical findings —normal.

Discussion

Cavernous sinus thrombophlebitis, characterised by swelling of the lids, chemosis, exophthalmos and fixation of the eye-ball may follow infection of the nose as well as those of the face and lips. The infection reaches the cavernous sinus by ophthalmic veins. The 3rd, 4th, and 6th cranial nerves which pass through the sinus are usually affected with motor disturbances. The infection occurs on one side but soon becomes bilateral within forty-eight hours. The history of the case under discussion is typical. He had boils on the right side of his face near the outer canthus. Subsequently, he developed swelling of lids, chemosis, proptosis with fixation of the eye-ball on one eye. Within forty-eight hours the same condition developed in the left eye with vomiting, headache and temperature. The history with clinical manifestations was characteristic. The condition which may present such symptom-syndrome is bilateral orbital cellulitis, or orbital phlegmon but bilaterality of such affections are most unlikely.

Summary

A case of thrombophlebitis of the cavernous sinus, which was hitherto an incurable condition, has been cured by combined systemic and intrathecal injection of penicillin. The administration of penicillin was mostly by intramuscular route.

Meningitis developed as a complication, which was successfully treated with penicillin and sulphathiazole therapy.

The penicillin treatment was supplemented by intravenous glucose 50 c.c. 25 per cent daily.

Acknowledgment

We are grateful to our chief, Lt. Col. P. Papatla, Professor of Ophthalmology and Oto-Rhino-Laryngology; Lt. Col. K. N. Pisharoty, Professor of Medicine and Dr. R. N. Sahu, Professor of Clinical Medicine for their kind advice and suggestions and to Dr. K. N. Misra, Superintendent, S. C. B. Medical College Hospital for his kind permission to publish this case report.

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CALCUTTA, DECEMBER, 1951

HAEMOLYTIC CONDITIONS

Intravascular hæmolysis is not necessarily a pathological process. In the normal phase-cycle of the red cells circulating in the blood, lysis and destruction take place. It has been estimated that in each second about 7 to 10 million red blood corpuscles are destroyed in the body.[1] This does not result in anæmia as the process is normally counterbalanced by the erythropoietic mechanism. Manifest hæmoglobinæmia also does not occur as the liberated hæmoglobin is promptly converted into bile pigments. The physiological processes involved in this hæmolytic mechanism are ageing of the corpuscles, spherocytosis, friction, phagocytosis and the action of hæmolysins present in the body.

When the degree of hæmolysis is considerably in excess of normal, hæmoglobinæmia results, but hæmoglobinuria does not occur unless the renal threshold for hæmoglobin which is 0·7 gm. per 100 ml. is exceeded. Excessive intravascular hæmolysis gives rise to methhæmalbuminæmia as well.[2] Hæmoglobinæmia is compensated by nature by excessive activity of the reticuloendothehial system, the effect of which is an overproduction of bile pigments and an increase in the icterus index of the blood.

Pathological intravascular hæmolysis may be due to corpuscular or extracorpuscular causes, and the cause varies from case to case.

Hæmolytic states may be of diverse ætiology. Severe burns and infective states such as malaria and infection with *Clostridium welchii* usually produce excessive hæmolysis. The various bacteria which possess hæmolytic properties *in vitro* seldom exhibit such propensities *in vivo*, but may occasionally do so. An example of this may be seen in the anaerobic hæmolytic streptococcal infection in puerperal septicæmia.

Toxic agents capable of producing hæmolysis in clinical practice are, snake venom, sulphonamides, phenylhydrazine used in the treatment of polycythæmia vera, benzedrine, potassium chlorate, phenothiazone in the treatment of threadworms, certain arsenical compounds, naphthalene, pamaquin specially when given in combination with mepacrine and lead. Fortunately, hæmolysis seldom occurs unless the individual is sensitive to these drugs.

Apart from the extrinsic factors mentioned above various intrinsic conditions may also give rise to intravascular hæmolysis. Defect in the structure of the corpuscles such as is present in congenital acholuric jaundice, sickle-celled and Mediterranean anæmias increase the fragility of the cells and promote their destruction during circulation. On the other hand, antibodies circulating in the plasma may lyse the red cells of the individual, or red cells transfused into him. Such antibodies are not always detectable by simple tests as they may not be of the nature of iso-agglutinins of Anti, -A, -B, -Rh, -Hr, varieties, but complicated cold agglutinins, cold hæmolysins and blocking antibodies which form a coating on the surface of red cells. Special tests may be necessary to detect these *e.g.*, the Donath Landsteiner test for the detection of cold hæmolysins and the Coomb's test for the detection of blocking antibodies. Thus, the extracorpuscular causes may act either as direct lysins or as immune antibodies which alter the red cells in such a manner that they are lysed in the presence of a complement. In other instances these agglutinins may agglutinate the cells and render them more susceptible to mechanical stresses or the hæmolytic mechanism is initiated by a change in the shape of red cells and occurrence of spherocytosis.

Hæmolytic anæmia may accompany or complicate such disease as chronic lymphatic leukæmia, carcinoma, lymphosarcoma, ovarian tumours, and Hodgkin's disease.

In recent years the frequency with which blood transfusions have been employed for therapeutic purposes has demonstrated the risk of hæmolysis attended with incompatible transfusions. The original tests based on the blood groups of Landsteiner have now been found to be inadequate. The hazards of Rhesus factor incompatibility, specially in those who have had previous transfusion and in women who have borne children, are being increasingly appreciated. The knowledge acquired in recent years has led to greater care being exercised in determining the compatibility between the donor's and recipient's blood before a transfusion is undertaken. From the point of view of compatibility Bird[3] had presented a useful summary of the routine tests that should be undertaken before a transfusion. In this country hæmolytic conditions met with in clinical practice are most commonly caused by malaria and blackwater fever. Blood transfusion, snake bite, and burns occasionally produce excessive hæmolysis. The other varieties appear to be rare.

Various hypotheses incriminating the malaria parasites, red blood cells, and circulating antibodies of diverse nature, have been used to explain the hæmolysis of blackwater fever. The subject is ably reviewed in the monograph on pathological processes in malaria and blackwater fever by Maegraith.[4] The evidences as to the cause of hæmolysis in these conditions are conflict-

[1] Best C. H. and Taylor N. B.—Physiological Basis of Medical Practice 5th Edn., 1950, p. 72.
[2] Fairley N. H.—*Quart. J. Med.*, 10: 95, 1941.
[3] Bird G. W. G.—*J. Indian M. A.*, 21: 67, 1951.
[4] Maegraith B.—Pathological Processes in Malaria and Blackwater Fever, Oxford 1948.

ing and the present state of our knowledge about the mechanism of hæmolysis in blackwater fever is incomplete. The communication on this subject in the present issue of the Journal by De and others[5], is welcome as an indication for increasing interest in the problem among the practitioners of this country.

Apart from hæmolysis which is a feature of blackwater fever, blood transfusion is believed to carry a special risk in this disease owing to the possibility of hæmolysis of transfused cells. Maegraith (*loc. cit.*) recommends transfusion when red cell count falls to 1·5 million per cmm. or below and emphasises the necessity of great care in matching.

The case reported illustrates the dilemma which is often faced by the clinician when treating a case of blackwater fever. He is tempted to use transfusion as a measure of therapy to save the life of the patient and yet in spite of repeated and energetic transfusions the condition of the patient may steadily deteriorate. In the case reported it is stated that group "O" blood was used in all four transfusions. Information as to what tests were done in order to determine compatibility are not given. It is desirable that in such cases all possible compatability tests should be made, specially when the transfusion is not done as an emergency measure. The biological incompatibility test devised by Weiner[6] cannot unfortunately be applied as an additional safeguard in cases of blackwater fever as hæmoglobinæmia is already present.

In the case reported the first transfusion was done when the hæmoglobin level was 57 per cent (Sahli). The red cell counts before the transfusions and during the various phases of the patient's illness are not stated. It is presumed that adequate tests were done as it was not an emergency transfusion. The necessity of such tests is all the greater as the patient had another attack of blackwater fever nine years prior to his present illness and it is not stated whether he had a transfusion then. Had it been so, the Rhesus compatability tests would also have been indicated.

Apparently, the excessive hæmolysis stopped at some stage of the disease before death ensued as the patient passed urine of 'almost normal colour' on the day of his death. A sternal puncture was indicated at this stage. Marrow biopsy could have demonstrated whether death was caused by an arrest of erythropoiesis. Owren[7] has demonstrated that "acute hæmolytic crises" of congenital hæmolytic jaundice, is attended with an arrest of erythropoietic activity of bone marrow.

Hæmolysis in blackwater fever continues to remain an interesting subject with wide possibilities of research. It is expected that pathologists and clinicians in this country would jointly undertake thorough investigations in a planned manner.

[5] De, S. N. *et al*—J. Indian M. A., 21: 95, 1951.
[6] Weiner A. S. *et. al—Am. J. Clin. Path.*, 12: 241, 1942
[7] Owren P. A.—*Blood*, 3: 231, 1948.

ROBERTSON MEDICAL SCHOOL, NAGPUR

We were surprised to learn from a recent order of the Government of Madhya Pradesh that the products of the newly created three years' course of training as medical and health assistants at the Robertson Medical School, Nagpur, would be recognised as full-fledged registered medical practitioners under the Madhya Pradesh Medical Registration Act on their engaging themselves in execution of the work entrusted to them for a period of three years.

We do not know if this was thrown out as a sop to the students who, of late, being convinced of their doubtful future began to agitate for specific diplomas and proper recognition.

Our Association did raise its voice of protest and unequivocal resentment when the Madhya Pradesh Government wanted to introduce the three years' course for training of medical and health personnel for the rural population as it was against our accepted principle of bringing about uniformity in the sphere of medical education which had been achieved by the abolition of the four-year medical courses and by the upgrading of some of the medical schools into medical colleges under the universities. The Government of Madhya Pradesh then assured our Association that those who would complete the newly started three years' course would not be given the status of doctors but would work in the rural areas as so many auxiliaries to the health and medical personnel.

We are really shocked to learn of the latest decision of the Madhya Pradesh Government which has gone back upon its own promise and ordered the acceptance of the three years' course for registration. We are positive that none of the other provincial Medical Councils would agree to accept this by way of reciprocity.

We are further disturbed to learn, if our information is correct, that the Madhya Pradesh Provincial Medical Council's opinion was not obtained prior to the issue of the aforesaid order. This is hardly in conformity with the existing practice all over India and is against the provisions of the law.

We are afraid, the decision of the Madhya Pradesh Government has added further complications in the problems that face the profession in this country. The Indian Medical Association at its Working Committee meeting held at Gauhati in October expressed its disapproval and deep concern over the matter and has addressed the State and Central Governments giving out its feeling on such a matter like this. The State Government order has confirmed the apprehensions we had when this dubious course was started, that some day these persons would try to get recognition as doctors and bring in undesirable complications.

Let us hope good sense will prevail upon the State Government and they will act in conformity with the scientific and civilised opinion accepted all over the country.

CURRENT MEDICAL LITERATURE

Etiology of Rheumatoid Arthritis

Baner and others (*Practitioner*, 166:5, 1951) write that rheumatoid arthritis is essentially an inflammatory disease in which neither the inciting agent nor the pathogenic mechanism is known. The inflammatory process could be initiated by a micro-organism, a harmful metabolite, a damaging exogenous agent, a metabolic degradation of one of the connective tissue components or the production of a defective connective tissue component. The complexity of the inflammatory response may be accentuated by local secondary reactions and perhaps by more remote metabolic phenomena. It seems apparent that the nature and the severity of the inflammatory process can be influenced within the individual by a mechanism which at least may be related in part to the pituitary-adrenal system. Whether some of the systemic manifestations of rheumatoid arthritis can evolve through this or other endocrine systems, particularly pigmentation, cessation of growth and alteration of menses, and a decreased excretion of 17-ketosteroids, is not known. There is no evidence that hypofunction of the adrenals plays a part in initiating the inflammatory process of rheumatoid arthritis or that adrenal hormones can completely suppress it. The adrenocorticotrophic hormone or cortisone-induced remissions of rheumatoid arthritis do not seem to be specific, for these hormones play a similar role in other inflammatory diseases, such as beryllium poisoning and certain hypersensitivity states.

Genetic and Endocrine Aspect of Gout

Smyth (*Practitioner*, 166: 62, 1951) gives in the following lines the summary of his observations:

Clinical gout occurs in those persons predisposed by hereditary hyperuricæmia.

A single, dominant autosomal gene is probably responsible for this transmission.

Recent reports clearly indicate that abnormal pituitary adrenocortical function is associated with the causal mechanism of gout. (*a*) Males have higher normal uric acid values than females, and this sex difference is usually not manifested until puberty in the male, and the menopause in the female. (*b*) Gouty persons have a low output of 17-ketosteroids in the urine. (*c*) The adrenocorticotrophic hormone (ACTH) of the pituitary, and cortisone are capable of both terminating and eliciting acute gouty arthritis.

There is an impressive body of evidence to indicate that final answers to the mystery of the etiology of this ancient disease may be found in the already heavily burdened adrenal cortex.

Adrenocorticotrophic hormone (ACTH) combined with colchicine is recommended in the treatment of acute attacks of gouty arthritis. The schedule recommended is as follows:

Colchicine and ACTH are begun simultaneously and given at six-hour intervals until 75 per cent to 95 per cent improvement is attained. The dose of ACTH is 50 mgm. given intramuscularly, and colchicine, 1/100 grain (0·65 mgm.) given by mouth.

When 75 per cent to 90 per cent improvement has been obtained continue to give colchicine, 1/100 grain (0·65 mgm.) orally, five times per day until the patient develops diarrhœa.

At this time, discontinue the colchicine for twenty-four hours and treat the diarrhœa, if necessary, with paregoric and bismuth preparations.

After twenty-four hours, resume colchicine, 1/100 grain (0·65 mgm.) three times per day, and reduce the interval to twice per day if gastrointestinal symptoms again occur.

When the maximum tolerated dose has been found, continue to give this ration for at least two weeks.

A.C.T.H. and Cortisone in Disease

Browne (*Brit. M. J.*, 1: 880, 1951) under the auspices of the Society for Endocrinology in his lecture on the subject first expounded the relationship of the general adaptation syndrome, as elaborated by Selye, to the action of these substances. He showed how the excretion of urinary corticoids was increased by a stress such as a long route-march; how it rose in pneumonia and after operations, only to fall with recovery. In pregnancy, too, the urinary corticoids rose, there being two peaks, one at about the sixtieth day and the second at about the seventh month; the values reached were similar to those seen in Cushing's syndrome, and Dr. Browne thought that pregnancy could be regarded as a physiological Cushing's syndrome. He cited another case where a sudden emotional stress caused a dramatic rise in the corticoid excretion.

Effects on Nitrogen Balance.—Acute trauma of all sorts, it was well-known, caused nitrogen loss—the loss coinciding with the increased adrenocortical activity. But long-continued stress was accompanied by a fall in corticoid excretion, and a negative nitrogen balance was changed into a positive one. These findings had led Drs. Albright and Browne to elaborate the hypothesis that a rise in corticoid production was accompanied by increased protein destruction. Discrepancies, however, soon appeared, and it had become necessary to modify this view. Dr. Browne now believed that in general the cortical steroids made the body protein more readily mobilized, and what happened to the protein depended upon the state of the body—in acute trauma, the protein was deaminated and lost; in pregnancy, it passed into the highly anabolic area of the uterus and fœtus and was retained. He believed also that there was a hierarchy of tissue requirements for protein—which might change in different circumstances. The effects depended on the state of the organism as a whole, and to illustrate this point he quoted the metaphor, suggested by Selye, of opening the window of a room—if it was hotter outside, the room got warmer, but if it was colder outside, the room got colder. The variability of the effects of cortisone, which could be explained on this view, were well shown by the failure of wound healing while under A.C.T.H. treatment in one case, but its normal occurrence in similar circumstances in another.

Dr. Browne then skilfully developed the thesis that A.C.T.H. and cortisone did not merely mobilize protein; they also caused shifts in electrolytes (sodium retention, potassium loss), water, vitamin C, histamine, the erythrocyte maturing factor, and so on. He described the effects of these hormones in a wide variety of diseases, showing how they could improve nephrosis, scurvy, pernicious anæmia—though he emphasized that he was not suggesting A.C.T.H. as treatment for these last two diseases—chronic pulmonary fibroses, asthma, etc. Remissions, however, were of variable duration and relapses usually occurred sooner or later. He pointed out the un-

toward psychological reactions which might be encountered and said he thought that A.C.T.H. merely brought out what was already there. By having the patient draw a male and a female figure at varying stages in treatment, fascinating changes were produced, coinciding with the patient's mental state—to the great amusement of the audience.

Cortisone and Tuberculosis.—The use of cortisone in pulmonary tuberculosis demanded the greatest caution; while under treatment, the patient usually seemed much improved, but the walling off of the lesion might be undermined so that on stopping treatment dangerous, or even fatal, exacerbation might follow. However, Dr. Browne described two cases of Addison's disease who had pulmonary tuberculosis and whom, because of the grave outlook, it was felt justified to treat with cortisone, streptomycin and P.A.S. One, who had developed crippling contractures of the legs under previous D.C.A. treatment—a very rare complication—gained 50 lb. (22·7 kg.) in weight, and showed all-round improvement. Her tuberculosis cleared, as did the pigmentation. In the other case general improvement also occurred in the Addisonian symptoms, but the pulmonary lesion did not change. But unless there were some serious complication, such as in the two cases above, he thought it hazardous to use A.C.T.H. or cortisone in tuberculous patient.

Finally, Dr. Browne drew attention to the fact that these hormones masked the symptoms of disease and obscured the criteria by which we were accustomed to estimate the severity of illness. He did not think they could cure many diseases, but they altered the body's reaction to them. They might exert a protective effect in some acute conditions—as in rheumatic fever, for example. He drew the picture of disease in general as an iceberg, with one-eighth above water—the observed specific reactions of the body to disease—and seven-eighths below water—the general reaction of the body. A.C.T.H. melted away some of the underwater portion, and caused the peaks to disappear below the surface—temporarily and sometimes intermittently. There was, in fact, a vast common underground of many different diseases; this it was that A.C.T.H. and cortisone affected, and this explained their apparent beneficial effects in such widely different disease conditions. He also emphasized the possible importance of emotional stresses in influencing disease, since it had been shown that such stresses could liberate adrenal cortical hormones within the body.

Salicylates and Cortisone in Rheumatic Diseases

In rheumatic fever the effect of salicylates on the pain and body temperature may be as great as that observed with 'Cortisone,' and it is tempting to suppose that the action of the two drugs is basically the same. Both drugs can reduce the temperature and blood-sedimentation rate, and both increase uric-acid excretion. Furthermore in the acute stage of rheumatic fever cortisone causes water retention followed by diuresis (Hench *et al.*—*Arth. Int. Med.*, 85: 545, 1950), and salicylates are said to have the same effect (Reid *et al.*,—*Quart. J. Med.*, 19: 1, 1950). Salicylates may also raise the fasting blood-sugar level (Morris and Graham—*Arch. Dis. Child.*, 6: 273, 1931) and cause acne Coombs *et al.*—*Proc. Cen. Soc. Clin. Res.*, 17: 27, 1944); and according to Reid and his co-workers (*Brit. M. J.*, 2: 1411, 1950) the increase in body hair and abnormal fat distribution as well as the reduced glucose tolerance and tendency to acne that are observed during salicylate treatment of rheumatic fever, are a mild form of Cushing's syndrome.

At the Royal Society of Medicine on March 13, Dr. Reid reported his results with a new salicylate-like compound—sodium γ-resorcylate—which differs from sodium salicylate in having an extra hydroxyl group in the 6 position of the benzene ring. In a preliminary trial of this substance, 7 cases of rheumatic fever responded rapidly with subsidence of the acute symptoms, although 1 patient developed a pericardial rub and effusion while still under treatment. The dose of the new compound is much less than that of sodium salicylate, and in animals its toxicity is less. Reid points out, however, that this substance, like salicylate can produce signs of mild Cushing's syndrome. At the same meeting Prof. G. A. H. Buttle remarked that γ-resorcylate is almost as effective as adrenocorticotrophic hormone (A.C.T.H.) in preventing the swelling that accompanies the so-called "formalin arthritis" in mice; and Dr. B. E. Clayton observed that, judged by standard skin wounds in mice, γ-resorcylate—and, to a lesser extent, salicylate—acts like cortisone in inhibiting formation of granulation tissue. Unlike cortisone, however, the new drug does not prolong the survival of mice with streptococcal septicæmia; and Sayer's test (ascorbic-acid depletion in the adrenals) has shown that it has no adrenocorticotrophic activity in mice. Dr. Smith, reporting an extension of his earlier work (*Lancet*, 1: 236, 1951), said that high serum levels of salicylate or of gentisate (which is another substituted salicylate compound with reputed anti-rheumatic effects) have no effect on the circulating eosinophils in man. In this issue Dr. Meade and Dr. Smith set out these latest findings, which suggest that salicylate does not have an A.C.T.H. like effect. Also in this issue, Van Cauwenberge and Heusghem show that in certain rheumatic diseases salicylates influence steroid metabolism. During salicylate therapy urinary excretion of reducing steroids was consistently and significantly increased; but there was no consistent change in the excretion of 17-ketosteroids.

Somerville *et al.*—(*Lancet*, 1: 116, 1951) have shown that steroid metabolism is abnormal in rheumatoid arthritis.

This collection of unconnected and partly unconfirmed findings affords at least prima-facie evidence of a relation between salicylates and cortisone. There are three possibilities. Firstly, the drugs may act quite independently but have the same final common pathway in the diseased tissues. Secondly, salicylates may have an adrenocorticotrophic action, although the balance of evidence is against this. Thirdly, salicylates may potentiate the naturally occurring hormone, either by blocking its destruction (as happens in hepatitis) or by increasing local tissue sensitivity—in rheumatoid arthritis cortisone is said to be potentiated by para-aminobenzoic acid (Weisel *et al.*—*Brooklyn Hosp. J.*, 8: 148, 1950). These hypotheses seem to be susceptible to proof or disproof by animal or clinical experiment. With recovery from rheumatic fever, however, biochemical changes may reflect the patient's clinical state rather than the therapy he has received. Hence, whether cortisone or salicylate has been administered, many changes may be the same; and one cannot infer from such similarity that salicylate produces its effects through the pituitary-adrenal axis. The action of salicylate and cortisone remain, nevertheless, two of the most promising "leads" in research on rheumatic fever; further work will no doubt establish their true relationship, and may yield some information about the nature of the local tissue changes. —*Ed. Lancet*, 1: 780, 1951.

Combined Salicylate and P.A.B.A. in Rheumatoid Arthritis

Hollander and Harris (*Am. J. M. Sc.*, 221: 398, 1951) give in the following lines the summary of their observations:

Experiments on 27 patients with rheumatoid arthritis confirm earlier observations by others that the administration of a combination of sodium salicylate with para-aminobenzoic acid produces a higher blood salicylate level than with an equal amount of salicylate used alone.

No change in the antihyaluronidase titer could be detected from the administration of salicylates alone or in combination with para-aminobenzoic acid.

From a clinical standpoint, the combination of salicylate and para-aminobenzoic acid seems to hold little advantage over salicylates alone in the treatment of rheumatoid arthritis.

Neurological Complications of Rabies Vaccine

Latimer and others (*Arch. Neurol. and Psychiat.*, 65: 16, 1951) report two cases of neuroparalytic accident after rabies vaccine. In case 1 the condition was myelitic in type, with the appearance of thoracic transverse myelitis after the tenth inoculation and subsequent recovery. In case 2 it was encephalitic in character, with a systemic reaction following the first injection and the development of encephalomyelitis with intercostal paralysis after the eighth inoculation the process terminating in death within 24 hours. Pathological changes included adventitial infiltration of cerebral vessels with round cells, perivascular microglial reaction and infiltration of the vagus nerve with segmented granulocytes (polymorphonuclear leucocytes).

Kala-azar Complement Fixation Test

Chang and Chang (*Chinese M. J.*, 69: 3, 1951) write that active antigens suitable for kala-azar complement fixation test were prepared from the infected human spleen as well as from the spleen and liver of hamsters and moles heavily infected with *Leishmania donovani*.

The sera of 112 proven cases of kala-azar and those of 83 non-kala-azar controls with various diseases as well as normals were subjected to this test, which was found to be highly specific, at least, in a diagnostic sense. The sera of 111 out of 112 or 99·1 per cent of kala-azar cases gave clear cut positive reaction. Those of the 83 controls all gave a uniformly negative reaction.

The test has proved entirely satisfactory and useful in the diagnosis of kala-azar, particularly in its early stage. It also has a prognostic value in the follow up of the treated cases and promises to be useful in the field survey of kala-azar.

Arthritis Due to Intestinal Amoebiasis

Rappaport and others (*Ann. Int. Med.*, 34: 1224, 1951) give in the following lines the summary of their observations:

Four cases of recurrent polyarthritis secondary to chronic intestinal amoœbiasis are reported.

Amœbic arthritis is clinically indistinguishable from rheumatoid arthritis and is subject to spontaneous remissions and exacerbations.

In all cases, gastrointestinal symptoms were minimal and arthritic complaints predominated. One patient never had diarrhœa, while in another arthritis occurred two years after the last attack of diarrhœa.

The diagnosis of amœbic arthritis was established by the isolation of the amœba from the stools and by the subsequent rapid response of the arthritis to amœbicidal therapy.

It is suggested that arthritis in these cases was due to sensitization to a toxin elaborated by the amœba, rather than to secondary infection of intestinal ulcers.

While amœbic arthritis is probably rare, examination of the stools for *Ent. histolytica* is indicated in all cases of 'rheumatoid' arthritis with concomitant gastrointestinal disturbances.

Drugs in Amoebiasis

The ideal amœbicide should be active by mouth; it should kill the organism in the trophozoite or cystic stages in the lumen of the gut, and should reach the liver and other infected tissues in a concentration high enough to deal effectively with trophozoites; and its toxicity must be low. No known drug satisfies all these criteria. The amœbicides now in use are of three classes. First, there are those like emetine, carbarsone, and 'Vioform' which when given systemically will kill amœbæ in either bowel or tissues; they presumably act directly on the amœba, since they have little, if any, effect on associated bacteria or protozoa. The second class act indirectly by rendering the bacterial flora of the intestine unfavourable for amœbæ; these include penicillin and some other antibiotics (probably 'Aureomycin' acts directly), sulphonamides, and hydroxyquinolines like chiniofon and 'Diodoquin.' These may clear the lumen of the bowel of *Ent. histolytica* but are not effective if the tissues have been invaded. Unfortunately they only clear the bowel of the parasites for a few weeks or possibly months, and when the normal bacterial flora returns the pathogenic amœbæ flourish once more. The third class of drug, exemplified by chloroquine, are concentrated in the liver and act well in amœbic hepatitis, but much less on the amœbæ inside the colon. For this reason a combination of amœbicides is often more effective than any one alone. Improved methods of *in-vitro* testing have made it possible to see whether an amœbicide acts directly on the amœba or on commensal organisms in the gut.

Several workers have been studying the effect of antibiotics in amœbic dysentery. Penicillin is used only as an adjunct to other treatment (Wright and Coombes—*Lancet*, 1: 943, 1948), since its therapeutic effect must be on the secondary bacterial invaders rather than on the amœba itself, which is resistant *in vitro* to concentrations of penicillin as high as 10,000 units per ml. (Knoll and Howell—*Arch. Path.*, 42: 594, 1946). Faust noted that 'Bacitracin' is amœbicidal and Möst (*Bull N. Y. Acad. Med.*, 25: 717, 1949) found that this antibiotic not only ends acute amœbic dysentery but also eliminates the amœbæ from the stools of two-thirds of the patients treated. It apparently acts selectively on clostridia and enterococci, as well as on other gram-positive organisms, but it does not prevent the subsequent reappearance of amœbæ in the stools; 80,000 units by mouth daily for 10 days is recommended. Aureomycin seems to be even more promising. McVay and colleagues (*South M. J.*, 43: 308, 1950) treated 37 cases with 2 gm. daily for a week with good results in 36; symptoms were quickly relieved and

the carrier state eliminated. Most *et al.* (*Am. J. Trop. Med.*, 30: 491, 1950) treated 100 cases with satisfactory results, though 10 per cent relapsed after 10 days' treatment. Armstrong and others (*Lancet*, 2: 10, 1950), working in the Amœbiasis Research Unit at Durban, obtained an immediate cure-rate of 94 per cent—higher than with any other single agent—but the relapse-rate was high. In their view aureomycin is the drug of choice in acute ulcerative amœbiasis, but it should be given with other drugs, such as diodoquin, to prevent the relapses commonly seen in the first month after a course of aureomycin alone. Hall (*New England J. Med.*, 244: 496, 1951), who has been treating acute and chronic cases in veterans at Minneapolis, reports good results in chronic amœbic dysentery and in cyst-passers (*i.e.*, "carriers," now usually looked on as chronic cases of amœbiasis); but he emphasises that "aureomycin should not be used alone in hepatic amœbiasis, but should be supplemented by emetine hydrochloride or chloroquine, or both." In his hands, chloramphenicol has been less effective than aureomycin against amœbæ *in vitro*. Thompson *et al.* (*Am. J. Trop. Med.*, 30: 203, 1950) found that chloramphenicol would suppress amœbic colitis in dogs, but only temporarily; it worked better against cæcal infections in rats but had not yet been tried in human infection. The amœbicidal activity of some of the new antibiotics have been compared by Watt and Vandegift (*J. Lab. Clin. Med.*, 36: 741, 1950). They head the list with polymyxin B and aureomycin followed by polymysin E. Bargen (*J. A. M. A.*, 145: 785, 1951) cites some excellent clinical results with 'Terramycin'; and lately McCowen *et al.* (*Science*, 113: 202, 1951) have shown that 'Fumagillin' or H_3, an antibiotic obtained from a species of aspergillus, will inhibit the growth of *Ent. histolytica* at a dilution of 1 in 120 million in the presence of the intestinal bacterial flora, and will eradicate amœbic infection in rats in a dosage of 9 mgm. per Kg. If this newest recruit is well tolerated by man it may well be the best amœbicide among the antibiotics.

Among other new drugs introduced for amœbiasis are 'Milibis' and chloroquine. Milibis, or 'Wia,' the bismuth derivative of p-N-glycolylarsanilic acid, originally studied in infected kittens in Germany in 1943, has been submitted to clinical trial in the United States by Dennis, Berberian, and Hansen (*Am. J. Trop. Med.*, 29: 683, 1949). It is not absorbed and so can only act locally in the bowel. Sixty-two out of sixty-eight cases were cleared of amœbæ after taking 0·5 gm. three times a day for a week, but further investigation is needed. Chloroquine [7-chloro-4-(4-diethylamino-1-methyl butyl-amino) quinoline] has given encouraging results as an amœbicide (Berberian—*J. Clin Invest.*, 27: 525, 1948). It is concentrated in the liver, the concentration in that organ being 400 to 600 times that in the plasma (Comen—*Am. J. Trop. Med.*, 29: 683, 1948). A dose of 0·3 gm. twice daily for two days and then 0·3 gm. daily for an additional twelve to nineteen days is a valuable adjunct in the treatment of amœbiasis with liver involvement. Chloroquine, despite its somewhat lower effectiveness in intestinal amœbiasis, is a satisfactory substitute for emetine in hepatic amœbiasis, and is considerably less toxic than emetine: apart from mild gastric disturbances chloroquine appears to be relatively free from side-effects. Emetine is still favoured for the treatment of amœbic hepatitis, but care is required in its administration since in dosage slightly in excess of that used therapeutically it is a protoplasmic poison. If the claims for chloroquine stand the test of time this drug may eventually replace emetine in the treatment of amœbic hepatitis.

Carbarsone, a trivalent arsenical compound, is often used in conjunction with emetine. If the oxygen attached to the arsenic atom of carbarsone oxide is replaced by a sulphydryl group (SH) the toxicity is reduced. Substitution of the hydrogen atom of the SH group by methyl or phenyl groups, and carboxylation of these to render the compounds soluble, results in amœbicides with an activity greater than that of the parent substance. After their administration arsenic can be detected in the bile, liver, kidneys, and intestinal tract. Preliminary tests in man have shown that two of these compounds are about 90 per cent effective against trophozoites in tissues and cysts and in the lumen of the colon. All arsenicals are, however, potentially toxic, and such compounds cannot compare in safety with antibiotics such as aureomycin and terramycin.

The problem of relapse may be solved by using a combination of drugs. Armstrong, Wilmot, and Elsdon-Dew found a combination of penicillin and sulphasuxidine, followed by emetine and diodoquin, 95 per cent effective. They believe that the dose of emetine could be drastically reduced if it were used with aureomycin, and that the combination of diodoquin with aureomycin would reduce the relapse-rate still further.—*Ed. Lancet*, 1: 1163, 1951.

Effect of Aminophylline in Emphysema

Whitfield and others (*Lancet*, 1: 490, 1951) write:

The lung volume has been determined, and tidal air tracings made, before and after the oral administration of aminophylline, in 6 normal subjects and in 12 patients suffering from emphysema without bronchospasm.

The drug did not change the lung volume or spirographic pattern of the normal subjects.

A slight reduction in the total lung volume, functional residual air, and residual air followed administration of the drug in cases of emphysema examined in the sitting posture, but no change in lung volume was apparent in recumbency.

The tidal-air tracings of the emphysematous group were unaltered by the drug.

No reason was found to suppose that aminophylline, in the absence of bronchospasm, has any place in the treatment of emphysema.

Cast Syndrome

Dorph (*New England J. Med.*, 243: 440, 1950) writes that the 'cast syndrome' consists of persistent vomiting and dilatation of the stomach in a patient to whom a body cast has been fitted. In describing one case, a girl aged eighteen years, which proved fatal, the author states that apparently only four other cases have been recorded, three of which proved fatal. In his case, necropsy revealed dilatation of the stomach and of the duodenum up to the ligament of Treitz, where there was an abrupt reduction in the diameter of the intestine. No evidence could be found of an organic obstructive lesion. The conclusion drawn from this case, and the others recorded, is that the greatest care must be exercised in the treatment of patients in body casts who present the symptoms of persistent vomiting and gastric dilatation. This precaution consists of immediate gastric intubation, removal of the entire cast, if necessary, and strict regulation of body fluids as indicated from laboratory and clinical findings.

CURRENT TOPICS

SOCIAL SERVICE DEPARTMENT IN A HOSPITAL

Its Organization and Functions

Dr. (Miss) G. R. Banerjee of the Division of Medical Social Work, Tata Institute of Social Sciences, Bombay, writes:*

ITS ORGANIZATION AND FUNCTIONS.—The ideal of social service is based on the twofold belief that the 'fulness of life' is the birthright of every man, and that if an individual cannot attain it by himself it is the duty of others to help him to do so. There is no field of social welfare to which this belief has made a greater contribution than to that which concerns health. As health is one of the essentials of life without which nothing can be achieved, the cause of the sick naturally makes a great appeal to the public. Founded on the principle of service to the sick, poor hospitals have grown till they have become highly specialised modern institutions. Similarly, clinics and dispensaries have also come into being to aid the sick.

For many generations we have seen new methods being evolved for rendering help to suffering mankind. The recognition of man's responsibilty for his fellowman is certainly not new to our country. From time immemorial prophets like Gautama Buddha, Shankaracharya and others have preached and practised it. Humanitarian workers have tried to help the needy by providing better facilities for them in the form of asylums, hospitals, schools etc. In short, they have tried to do something *for* the client which *they thought* was best for him. However, much was still lacking in the understanding of human behaviour. Very little was known as to the type of assistance which would be most suitable in helping the client *to help himself* and the type of aid that would be detrimental to his welfare in the long run. Humanitarian work or what was called social work, until recently, was based upon the goodness of heart of the worker. If she felt that she *wanted to do* something for her fellow beings and thought that that something was good for them, then she started working towards that end. There was little thought that real social service needed a special training.

With increased knowledge of human behaviour in recent years emphasis is now being placed upon professional training for social work in countries like the U.S.A. and the U.K. The profession of social work to-day presupposes the acquisition of a scientific body of knowledge which should be acquired in a school of social work before any individual can be entitled to practise social work. In our country the term social service is still identified with humanitarian work of all sorts. In that broad sense of the term a doctor may be said to be rendering social service, as also an engineer, a teacher or a housewife. But anyone engaged in welfare work cannot be called a social worker to-day. The fact that a person is employed in a welfare agency does not by itself make him a social worker. It is the way in which he works with people, individuals, groups and communities, the skill he uses and the manner thereof that constitute the criteria as to whether or not the service he renders is "professional social service." Such a service is one in which professional skill implements man's deepest instincts in offering help to his fellows. In India, this professional social service which is based on a distinctive body of knowledge and skill is just in the process of development.

WHY HOSPITAL SOCIAL SERVICE? Hospital social service is a branch of professional social work and is oriented to medicine. It has arisen to fill gaps in the application of medical treatment to a patient. With our growing knowledge of man in his social aspects it has become increasingly evident that various professional skills in co-operation with medicine are necessary to make medical care adequate. It has now become obvious that although scientific discoveries may continuously advance medical treatment, the most expert care may be of little avail if the social and emotional components involved in an illness are ignored. Physicians and surgeons are often baffled by the reaction of patients to the treatment they recommend. For instance, a mother may refuse to agree to a surgical operation for her child even when it may be the best and the only method of cure; or a T.B. patient may refuse to undergo sanatorium treatment, or a V.D. patient may be irregular in his attendance at the clinic in spite of a warning as to the serious consequences of neglecting the disease in its early stages.

In the case of such patients it is necessary to consider the underlying causes of their peculiar reactions. The causes are so numerous that listing all of them is not possible. However, we can discuss a few of them. Let us take the case of a T.B. patient who resists being taken care of in a sanatorium. Such a person may have an intense fear of the possibility of additional infection from other patients, or the feeling that loss of activity will turn him into an invalid. Or again, treatment in a hospital or sanatorium, away from his family and society may mean not only being shut away from the near and dear ones but also being branded with a social stigma for the rest of his life. Sometimes a patient may regard his illness as a punishment for past sins, and often he is known to think that he will never be able to return to his family and community. Or take the case of children whose parents resist hospitalisation. Often mothers who may have rejected a child even before it was born, feel guilty when it is ill and reject any treatment which may be painful to it for the time being. They may try to overprotect their sick child, spoil it and make it dependent on them to make up for the secret feeling that they have been the cause of its illness.

These factors may seem ridiculous to us who are not affected by them but to those who are facing them, they are very real. Since patients themselves are not conscious of the factors that create these fears in them they are unable to get rid of them. Unless these are handled scientifically the patients will remain emotionally upset and unable to accept the prescribed treatment.

In this connection it might be mentioned that with the development of psychosomatic medicine dealing with the inter-relation of psychological and physiological aspects of disease, an impressive body of scientific evidence is being produced regarding the significance of emotional factors in a patient's illness. It has been found that many patients suffer from physical symptoms which may not necessarily have an organic basis or which may have both organic and emotional bases. There was a time when doctors felt that

* Based on a paper read at the All-India Medical Conference held at Sholapur in December 1950.

there was nothing wrong in such cases after the organic basis had been treated. With the gradual advancement of the psychosomatic approach in medicine, doctors are beginning to realise that the symptoms of emotional illness are not imaginary but painfully real. A knowledge of the physical and social environment of patients has been found invaluable in interpreting some predisposing and causative factors in illness.

Besides emotional factors there are various social factors that need to be tackled in taking proper care of the sick. Often a homeless patient comes to the hospital in an acute stage of illness; he is treated; and after he recovers, he goes back to the same environment. Naturally he returns again with the same acute symptoms. Or a patient may become a total invalid and incapable of doing any work by which he can earn his living and, therefore, ends up as a begger unless he is rehabilitated and helped to live a fairly normal life with his illness. Somebody needs to work with the patient not only at the time of diagnosis and during his period of hospitalisation but also long after his discharge. He may need readjustment to his home, or to his school life, or to some new kind of work. Thus a due consideration of social and emotional factors is absolutely essential if the hospital is not to become just a repair shop but a link in the chain between a period of broken health and one of health restored

It is true that the doctor is responsible for the care of the patient but, due to the advancement of medical science, specialisation rather than general training in medicine is becoming more and more the practice of the day. Perhaps this is essential for rendering expert medical care. Now-a-days a patient in a hospital can get the services of several specialists, each of whom will concentrate on the various aspects of his disease. Yet we know that disease is not a disembodied entity. It is not just the unhealthy throat or the leg that succumbs to the forces of pathology. It is always the personality as a whole that reacts; that is, it reacts in its social, emotional and somatic aspects. Unless specialists try to understand a patient as a distinct personality he may appear to them merely as a pair of lungs or kidneys lying between two bedsheets.

With specialisation coming into the medical field there is a drift away from the family physician who was generally familiar with the patient as a person. He knew the personality and the home background of his patient and, therefore, could see how these affected his diseased condition. To-day, however, since more and more hospitals are being established to give better medical aid to the sick, the medical profession has to deal with patients away from their home environment and relatives. This in itself may accentuate the emotional problems of patients, while it also prevents the physician from knowing his patient as a total person. As the specialist has a vast amount of work in the field of his specialisation, he is obliged to delegate certain aspects of his patients' problems to some other individual who is specially trained to handle them. This means a division of responsibility between the doctor and the social worker with their respective concentration upon the medical and psychosocial aspects of illness. Thus the need arises for creating a department of social service in the hospital, like the x-ray department, surgical department or gynæcological department to enhance the value of medical care.

Role of the Hospital Social Worker: Generally the value of Hospital Social Service is recognised for its ability to acquire financial aid when required for patients, as it keeps in close touch with such community resources as trusts and charities of which neither doctors nor patients may be aware. As for the help a social worker may render in other fields, the question is frequently asked, "What can a hospital social worker do about the social and emotional problems of the patient?" There is much that a hospital social worker can do for a patient besides securing him financial aid.

When a social worker trained in understanding the dynamics of human behaviour and the technique of interviewing, is attached to a hospital, she can interview at the point of diagnosis those patients who show resistance to the medical care prescribed for them. Through 'case work technique' as it is called, she can help the patient to realise and express the reasons why he feels the way he does. As a result he acquires an attitude of objectivity and reasonableness. Opportunities for expressing his partially repressed feelings to somebody who understands and accepts him as he is, may help to relieve him of his anxieties and provide a sense of support. This, in time, increases his ability to solve his problems. Carefully planned interviews, in most instances, can help the patient to give up the irrational attitude so commonly found in one who is in distress, accept the reality of his situation and act accordingly. The reaction of the patient, however, psychoneurotic it may seem to us, is in essence a reaction to some conflict or fear motivated largely by subconscious psychological processes of which the patient himself is totally unaware. Planning an interview in such a way as to bring to light significant factors is a service which can be rendered only by a trained social worker.

After helping the patient to accept his diagnosis and medical recommendations, the social worker keeps in close touch with the patient and his family. It is her duty to see that there are no obstacles in carrying out the treatment. If there are complications in the domestic relationship that come in the way of his treatment the social worker deals with them. For example a newly married husband who has not yet tested the strength of his marital bond may wonder what his wife will do when he is in hospital. He may not like to leave his home for treatment or if he is in hospital he may become more and more suspicious and finally demand to be discharged. A social worker understands that in such institutions latent anxieties are prone to emerge, and that many of these anxieties arise from a feeling of inadequacy, created by a long period of illness. Skilful interpretation, a positive relationship between the patient and the worker, and the co-operation of all the members of the family are means to help the patient rid himself of his worries.

Apart from helping the patient to adjust himself to his illness, the social worker also helps the family to adapt itself to the new situation caused by this illness. If for example, the patient has a wife who feels utterly helpless and finds the responsibility of the household overwhelming while he is away from home, the social worker gives constant moral support to the wife and helps her to assume her new position of responsibility. Thus by working with the patient and his family, the social worker lessens his anxieties and enables the patient to complete his medical treatment. She sees that he does not 'sign out' against medical advice.

Further, if necessary, a social worker may follow a case even after the patient's discharge from the hospital. If lasting advantage is to be gained from the treatment given in a hospital, there must also be a proper plan for 'after care,'

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2.83

A social worker may have to assist a dischargee in securing a job better suited to his physical condition and also get him reconciled to his new work.

Patients may have varying ability to face a disability caused by illness; but each one has a right to be regarded as an individual with hopes and fears and aspirations. Without knowledge of the patient as an individual it is not possible to open up for him any vistas broader than the illness with which he lives. The limitations of illness should not be overlooked but should be seen as part of the patient, affecting the whole of him but not obliterating him. Medical care to be effective requires consideration of the total needs of the individual—social and emotional as well as physical. While this requires that all persons responsible for medical care be concerned with the patient's social and emotional needs, it is the primary responsibility of the social worker to focus attention on such needs and to further the mobilization and integration of services to meet them. She brings to the doctor knowledge of the patient as an individual which facilitates planning his treatment as a person and not for his diseased condition alone. Hospital care planned on these lines usually shortens the duration of a patient's illness and consequently eliminates need for repetition of medical treatment.

Besides working directly with the patient the social worker can arouse in the members of her team (doctors, nurses, health visitors, occupational therapists and physiotherapists etc.,) an awareness of the social and emotional factors as they relate to medical and health needs or affect the provision of medical and health services for the patient. She can also stimulate consciousness of the said factors in the personnel formulating the programme of the hospital, in other agencies, organisations and individuals, interested in, or having a potential interest in the patient. The participation of the social worker in ward rounds, staff meetings and case demonstrations is of great value for this purpose.

A trained social worker in a hospital can be of immense assistance in medical research. Since she helps the patient to accept medical recommendations and works for the continuity of treatment, she can enable physicians to observe the results of any particular treatment in which they may be interested. Or, when a patient does not respond to a certain type of medical treatment, the social worker, who is familiar with his mental and social background, can find out for the physician whether or not the deterrent is a social factor which first needs handling before he can be expected to respond. Such information would be of great help in the verification of his reactions to a particular treatment. Without the aid of a social worker in a hospital, doctors would find it difficult to ascertain whether the response noticed in a patient is the direct result of a particular medical approach or of complications in his family situation. For example, take the case of a tuberculosis patient who has been under expert medical care for about a year without showing any improvement. One day the patient told the nurse that she had sleepless nights due to her worries. The nurse referred the case to the social worker who interviewed the patient. Thus the worker came to know that the delinquent behaviour of the patient's daughter had brought her to the attention of the juvenile court, and that the court had deprived the parents of further custody of the child, awarding her to a near relative of the father. This relative was withholding information about the girl from the mother. The social worker helped this patient to give vent to her feelings of anger and bitterness and thus provided her with the much needed opportunity of talking about her feelings of guilt for having failed as a good mother. Further, she made it possible for the patient and her daughter to correspond with each other which was mutually satisfactory. It re-established a constructive relationship between the two. After this, the patient started showing improvement in her condition.

A trained hospital social worker can promote social research. She has not only knowledge and skill in meeting the social and emotional needs of a given patient in so far as is possible, but also the capacity for seeing a case in relation to the common factors producing social problems. A social worker needs to carry an enquiring mind into every case. Her whole approach to a case and its handling is shaped by a twofold purpose to serve and to learn. The worker's mind must focus upon determining what is contributed by each case to the knowledge and understanding of human beings and social forces. She asks: What does this case have in common with other cases which can permit us to generalise on some common need and justify further study to determine the necessity for social measures? It is the responsibility of the trained social worker to convey the knowledge of this need to the public and to present her findings intelligently and convincingly so as to stimulate action. Properly kept records of cases handled by the hospital social worker can provide ample data for social research by others, which in turn can lead to social action for promoting the cause of the sick. The hospital social worker should also co-operate with other public and private agencies in developing new resources in the community and participate in its effort for the prevention of disease.

FUNCTIONS OF THE HOSPITAL SOCIAL WORKER:—The functions of a hospital social worker are various and variegated and can be listed briefly as follows:

A. *Practice of Social Case Work:—*

(1) Selection of those patients whose restoration to health can be expedited by the co-operation of the social worker with the physician.

(2) Study and evaluation of the social and emotional factors which reveal a patient's willingness to accept the diagnosis and recommendations for medical care, and his ability and capacity to undergo the prescribed treatment.

(3) Dealing with those social and emotional factors which contribute to a patient's resistance to the recommendations of the physician, or interfere with the patient's gaining full benefit from medical care.

This service can range from brief help within the hospital itself to a very comprehensive study and extended service. It may involve direct interviews with the patient and his relatives or visits to his home or both. Such a service might continue even after a patient is discharged medically.

B. *Interpretation of the significant social and emotional factors involved in a patient's illness to the physician concerned, for joint planning of medical and social care.*

C. *Referring cases to co-operating welfare agencies of the community when it is important for proper planning in the interest of the patient.*

D. *Social Orientation of the hospital staff:—*

Since the social worker brings to the medical setting knowledge and skill which is different from that of any other

group within the hospital, it is appropriate for the worker to arouse in the staff an awareness of the social and emotional factors that are related to the medical and health needs of the patient. This can be achieved by participation in ward rounds and staff meetings and also by a programme of in-service training.

E. *Participation in the education and training of student nurses, medical students and students in hospital social work etc.*

F. *Participation in the determination and formulation of hospital policies with a view to socialise the hospital set-up.*

G. *Participation in community planning for health and welfare.*

A hospital social worker must take up the responsibility of working with organised groups, and of stimulating others towards action in these areas. This may necessitate many public relations activities such as talks before community groups, participation in committee meetings and the like.

H. *Maintaining social work records:*

A hospital social worker should keep social work records in the social service department. This is important not only for the purpose of social statistics but also for better diagnosis of the social problems. It also acts as a good check for the social worker on the social treatment plan formulated by her. The nature of social case work is so complex and fluid that every factor must be very carefully described. The social worker must improve her skill constantly. She must be a student and a critic of what she does; and there is no more effective way of thinking than by writing down one's observations and reflections. The quality of performance is bound to be heightened when every act is carefully evaluated. Recording also helps in the acquisition of the habit of careful observation and accurate description in writing. Moreover, no worker can remember all the details of all her cases. Then too, if a worker takes leave for a few days or is ill in the course of her service, another social worker may be able to deal with a case in her absence, if all the details concerning it are already carefully recorded.

From time to time she should submit written reports about the social and emotional problems of some patients to the physician treating them. Such reports should not be written on the medical case sheet at all. It will be confusing if a report on the social and emotional factors is written below a medical report. Besides, the physician may not notice it when he is in a hurry. Therefore, a better procedure would be to use sheets of a particular size and colour, with 'Social Service Department' printed on top for writing social service reports for physicians. These report sheets should be attached to the medical chart. It may be mentioned here that social service reports for doctors should not be in such detail as those meant exclusively for social service department. On these coloured sheets only that portion of social information may be given which a physician may need to know for purposes of diagnosis, treatment and discharge plan of the patient. The language of the report must be simple. Technical terms used in case work practice have to be carefully avoided as physicians may not be familiar with them.

A social worker should also be particular about other details of recording *e.g.*, filling in the face sheet for each case. The chief purpose of this sheet is to give identification data about the persons most involved in the social situation. Details like name, address, occupation, relationship, nationality, religion, date of birth and social status (*i.e.*, whether single, married, widowed or deserted etc.,) distinguish a person and his family from others of the same name. Besides the columns on 'social service no.', 'ward no.', 'cot no.', 'Physician in charge,' 'referred by,' 'social worker' help to locate the patient administratively in the agency rather than in society at large.

For every social service case, a card index should be made out by the worker giving in a convenient form some tangible social facts of a more or less permanent character. The columns in the card should be so arranged as to make possible the entry of several changes of address and places of employment without the need for making a fresh card at frequent intervals. This card index should become the central file for subsequent identification.

A hospital social worker should regard it as her responsibility to see that social case records are kept in a systematic way in the department. All case records should be numbered and each of them must have a separate file bearing the name of the patient and his 'social service number.'

I. *Writing periodic reports of the social service department.*

J. *Taking up social research projects or co-operating with other agencies in their research programmes whenever needed.*

Qualifications and Salary of a Hospital Social Worker:—The Social Service Department in a hospital should have well qualified workers. No person should be allowed to take up a hospital social worker's post unless he or she has had the necessary specialised education in the field, maturity of judgment and a definite interest in hospital social work. Academic preparation for hospital social work is extremely valuable and essential. An ideal combination would comprise of (1) adequate background of general education, (2) interest in the sick (3) ability to overcome fear and aversion at the sight of blood, disease and death (4) special training in a school of social work in courses comprising of subjects, such as, Sociology; Social Pathology; Indian Social Problems; Social Legislation; Case Work; Group Work; Community Organisation; information on psychiatry, psychology; symptoms, causes and treatment of diseases; psychosomatic medicine; social and emotional components of illness and their care; and administration of social service department. Students should be familiar with research methods also. A hospital social work course *per se* is not sufficient. Every hospital social worker should have a general education equalling or exceeding a bachelor's degree while hospital social work training should be on a postgraduate level, accompanied by supervised field work placements in hospitals.

The salary of the hospital social worker should be in accordance with the training required for the position and the responsibility for the welfare of human beings which the position carries. The starting salary of a social worker with specialised training in medico-social field should in no case be lower than the starting salary of a medical man. When she becomes a senior social worker, her scale should be that of a senior medical officer. Besides, the hospital should meet all transportation charges for home visits or visits to community resources. The social worker should also be entitled to provident fund benefits or pension as the case may be in a particular hospital. Adequate salary coupled with high academic requirements should produce workers of integrity

imbued with the spirit of service and a self-imposed responsibility for continuous education in new developments.

Some Personnel Practices—

(1) *The Health Services of the hospital should be extended to its social workers.*

(2) *Social workers like other members of the hospital staff should be eligible for sick leave and paid vacations as well as Bank Holidays.*

(3) *Education leave*: To enable hospital social workers to keep in constant touch with new developments in their field of service they should be given education leave every few years to attend refresher courses or take some advanced training.

(4) *Hours of work*: The social service department should work on the basis of 5½ days a week, with seven hours per day, including an hour's break for lunch. People may ask why a hospital social worker should not work on an 8 hour day basis. The reason is that frequently she has to work late in the evening or early in the morning or at any odd hour of the day. Therefore, this one hour concession seems quite reasonable.

A social worker like all the other employees of the hospital should be required to sign the daily attendance register. It is absolutely essential that the social worker should come to the hospital daily though she cannot be required to be present on the hospital premises for all the six hours. She has to make home visits or visits to community organisations and the time spent on them should be deducted from the daily work period of six hours. Or again, a social worker may have to make a home visit late in the evening or early in the morning if certain clients are available only during those hours. These visits are bound to be outside her hospital duty hours.

The hospital social worker should keep a regular diary and write an account of the hours she devotes to her work. If she writes down her weekly programme, the hospital superintendent or her supervisor will be able to know the amount of time she spends on hospital work. On some days if she has to work a little more, due to the nature of her cases, she should be willing to do so. But she should be allowed a few hours off on other days to make up for these extra hours. Thus keeping a diary of her daily hours will facilitate her work as well as that of the supervisor who will then know what type of work the social worker is doing.

In the interest of the Social Service Department she should make it a point, if she is the only worker, not to leave the hospital during the Out Patient Department hours because many new patients may have to be seen at that time. If the Out Patient Department works both morning and afternoon then she should fix up certain mornings and afternoons for visits to patients ' homes or agencies ' offices.

(5) *Case load*: At no time should the case load of a worker be more than twentyfive. Of these, half should be active cases requiring intensive work while the rest should need only minor assistance. As cases get closed or become inactive for a period of time, new ones may be taken in their place.

FACILITIES—

(1) *It is desirable that every social worker should have an office room of her own.* If, due to shortage of space, it is not possible to give a room to each worker, then at least one big office room should be supplied to two or three social workers. This room should be fitted with interviewing booths where each social worker can interview her clients in privacy. Equipment for the office should include tables, chairs, book cases, filing cabinets, files for records, stationery, lamps and fans.

(2) *The Social Service Department should be so located as to have an easy access to telephone connections and clerical staff for secretarial help.* If there are three or four social workers in a hospital then there should be at least one clerk appointed for the department. But if there is only one social worker the secretarial work, like typing a few letters, etc., should be managed by the hospital clerk. *The same holds true of a peon* for the department.

(3) *Professional library*: This should be set up as soon as the department is established and new publications should be added from time to time.

(4) *Patients' social welfare fund*: It is of utmost importance that the Social Service Department should have an emergency welfare fund. Sometimes patient's health or even life is jeopardised by non-availability of financial assistance. In such cases, the fund will be of immense use to needy patients. As the social worker has received special training in the methods of dealing with people's financial needs, the welfare fund should be in her charge. The fund should form part of the Social Work Department budget.

ORGANISATION OF A HOSPITAL SOCIAL SERVICE DEPARTMENT:

(1) *As a professional unit of the hospital organisation the social service department should be organised on the same basis as other departments of the hospital.* It is essential to maintain centralised control irrespective of the sources of support. Outside help should never be rejected; it should rather be stimulated. The expenditure, however, should be controlled through the general administration of the institution.

(2) *The Director*: As hospital social work is new in India and as its training has only recently been started, it may take a few years before hospitals can have senior social workers (not only in years but in experience too) who can occupy the posts of directors of Social Service Departments. It is always better to employ only one or two social workers when a Social Service Department is first organised in a hospital. As the work of the Department grows, more workers may be taken in. All of them should be directly under the hospital superintendent. After about five or six years the worker who is most efficient should be made the director of the Social Service Department. The director should be responsible to the superintendent and have an equal status with other departmental heads and join them in the discussion of hospital policies.

(3) *Volunteer Service*: The Social Service Department should organise a volunteer service and work in

close co-operation with it. Such a service is not meant for duplicating the work done by the Social Service Department but to complement it. It will be helpful to have a volunteer service for getting various types of assistance *e.g.*, enlisting blood donors, collecting gifts for children, building up a library for patients, taking patients home and raising funds for the hospital. But this type of service should not be mixed up with medico-social work which requires specialised training.

(4) *Referral to the Social Service Department*: Ideally the majority of cases referred to the Social Service Department should come through the doctor but at present doctors may not be sure which cases they should refer to the Social Service Department. With a limited number of social workers in hospitals, a *via media* has to be struck. The social worker should interview all cases of T.B., major cases of surgery and cases of invalidism. From the other sections the social worker may take such cases as are referred to her by doctors and nurses. In those cases which she selects herself in the course of her interviews, she should consult the physician as early as possible. Work on individual cases should always be in association with the physician responsible.

Is Hospital Social Service Costly:—Some people may wonder if it is worthwhile for a hospital to spend money on Social Service Departments when there are many other needs to be met. While from the point of view of immediate needs it may seem an unjustifiable expenditure, in the long run it will be a paying proposition. A patient who is discharged without adequate planning for 'after-care' will have to be readmitted every time he develops acute symptoms. Consequently, he becomes a source of great expense to the hospital. Besides, it is a tremendous human waste too. A patient who is medically discharged from the hospital but lies in the ward, because there are no adequate plans made for him and he has no place to go, adds to the hospital's expense. A patient who refuses due to inner conflicts medical treatment at an early stage of his disease and comes when acutely sick also adds to the hospital's expenses. As already stated, a trained social worker can help the hospital in these problems and not only enable it to save its funds but also be of better service to the sick and thereby to the community.

However, it must be admitted that social service can prove very expensive if proper use is not made of it. The social worker should never be used for odd jobs which can easily be done by less qualified and less paid workers. This is frequently overlooked as hospital authorities are not always familiar with the functions of a professional hospital social worker. For example, she is sometimes allotted the task of writing letters to a patient's relations to come and take the discharged patient home. This is given to her just because the clerks have too much work to do. On other occasions she is asked to telephone some other hospital or infirmary for the transfer of a patient, which is actually the duty of the hospital clerk. She may also be asked to accompany a patient to the infirmary or to some other institution when the patient is being transferred from the hospital and taken in an ambulance. The reason given is that the hospital cannot spare an ayah or a ward boy to accompany him. Surely, in the long run it would be cheaper to have two clerks or one more ward boy to do these jobs than to have highly paid, trained social workers do them.

Rather than have hospital require its social worker to collect blood for its blood bank, it might be suggested that the hospital ask for volunteer service or employ a person with an aptitude for canvassing. It would be expensive for the hospital as well as for our country to use trained social workers for such a purpose or for bill collecting. Some hospital authorities think that social workers should undertake the task of directing patients from one department to another. It is true that most patients and their relatives are illiterate and do need such help, but would it not be less expensive for a hospital to appoint an untrained person for this type of work on a part time basis?

Doctors often think that a social worker should follow up every case discharged from the hospital. There are many cases of 'follow-up' where no case-work skill is needed. If doctors and nurses are sympathetic most of the patients respond voluntarily as a result of the favourable contact made in the clinic or hospital. However, there may be some cases where even when the patient likes to return to the out-door department regularly, she cannot do so on account of her home situation. For example, a woman who has young children will find it difficult to visit the clinic regularly if there is nobody to look after them during the time she is away. Therefore, if a part-time clerk is engaged to send out letters which the patient can have read to her, and if these letters have warmth and show interest in the patient, it is possible that she will let the clinic know about her difficulties and agree to the programme that the clinic may make for overcoming her handicaps. When the clinic clerk receives a reply, he will know the obstacles in the way of the patient and will refer the matter to the social service department. Thus, a great deal of the social service department's time will be saved by this method, which would not be possible if it has to follow up each case.

It is thus evident from all that has been stated above that a hospital social worker has a definite and distinct role to play in the programme of patient's treatment. Modern medicine and hospitals should consider the social worker as an important specialist who can contribute substantially to a total study of a patient and to a complete diagnosis. Only then will they be able to gauge the value of a hospital social worker and make the best use of her in the interest of the patient, the hospital and the community.

NOTES AND NEWS

THE INTERNATIONAL PHARMACOPOEIA

The first International Pharmacopœia setting up uniform world standards for 200 drugs in universal use was published recently by the World Health Organisation. It also establishes internationally-recognised Latin names which will avoid the existing confusion arising from the use of different names for the same drug.

Up to the present most countries have used their own independently-produced national pharmacopœias, often called "the pharmacist's bible," which lay down official drug standards to meet national requirements.

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It is expected that the universal adoption of the International Pharmacopœia, as formally recommended to WHO Member States by the World Health Assembly, will further the spread of medical knowledge, increase economy in drug production, and facilitate international trade in drugs.

The book published is the first volume of the International Pharmacopœia. It includes 43 appendices describing the procedures necessary to establish the purity and strength of the drugs dealt with, and also a note on patents and trademarks.

Volume II is in preparation by the WHO Expert Committee on the Unification of Pharmacopœias at present in session in Geneva under the Chairmanship of Dr. C. H. Hampshire of London. Volume II will include the newer synthetic remedies, antibiotics such as penicillin and streptomycin, and also specifications for the administration of drugs by injection, in tablet and in liquid form.

The International Pharmacopœia is appearing simultaneously in English and French, and a Spanish edition will follow.

The publication of the International Pharmacopœia is the result of the work of many experts undertaken since 1937 under the auspices of the Health Organisation of the League of Nations. The Expert Committee appointed by the WHO Interim Commission in 1947 is now bringing this work to a successful conclusion under Dr. Hampshire's chairmanship.

WHO PREPARES BIRTH CONTROL PLANS FOR INDIA

The World Health Organization is preparing an experimental birth control programme for India, it was revealed at San Francisco during the opening session of the American Public Health Association Convention.

Officials emphasized that the programme would not start until all details were considered and a "broad outline for experiment" was worked out and approved by the Government of India.

The Indian Government's request for WHO aid on its population problem was revealed by Dr. Chisholm, Director-General of the WHO. Details of the proposal were outlined by Dr. C. Mani, Regional Director of the WHO in S.-A. Asia with H.Q. at New Delhi.

In any birth control programme experiments sponsored by the WHO, the so-called "rhythm system," would be employed to meet most religious objections, it was stated.

Dr. Mani said that Dr. Stone of the Planned Parenthood Association of New York was serving as consultant to the WHO on the project and was now in New Delhi.

"Dr. Stone will explore the possibilities of carrying out some kind of scientific experiment to find out what the proper approach to this problem might be. He will stay a month or six weeks in India to study local possibilities and draw the broad outline of the experiment."

When Dr. Stone completed his work, Dr. Mani said, two experts would be sent to India from the U.S.A. They would attempt to put the provisions of the "broad outlines" into effect.

WHO WORLD ASSEMBLY VOTES IN NEW MEMBERS

During the recent Fourth World Health Assembly, at Geneva, three more nations were admitted to WHO membership. They are: Japan, the German Federal Republic and Spain. Formal admission to membership, however, entails the depositing of the nominated nation's acceptance of the WHO Constitution with the Office of the Secretary-General at United Nations Headquarters, a requirement which Japan fulfilled immediately.

Upon receipt of acceptances by the other two countries, WHO membership will have reached a total of 78. At present, there is one Associate Member.

In accordance with WHO policy, one-third of the 18-member Executive Board was elected also during the Assembly. Chosen to serve a three-year term were representatives from: Belgium, Ceylon, Cuba, Greece, Lebanon and Liberia to replace Brazil, India, Union of South Africa, Poland, Yugoslavia and The Netherlands.

Other Executive Board members for the ensuing year include the delegates from: Chile, El Salvador, France, Italy, Pakistan, Philippines, Sweden, Thailand, Turkey, United Kingdom, United States of America and Venezuela.

WITHDRAWAL OF QUARANTINE RESTRICTIONS

Information has been received by the Director-General of Health Services, New Delhi, that the Netherlands New Guinea and Indonesian health authorities have withdrawn quarantine restrictions on account of small-pox against passengers from Delhi.

The Netherlands New Guinea and Jordon health authorities have withdrawn quarantine restrictions on account of cholera against passengers from Bombay. The Philippines health authorities have also removed this restriction against passengers and crew who arrive at Manila from Bombay and other ports of India where there have been no case of cholera.

NO SALES TAX ON SKIAGRAMS

The Government of West Bengal have informed the Bengal Radiological Association that "Radiologists are not subjected to pay sales tax on skiagrams handed over to patients with their expert opinion." Also "Radiologists are not liable for registration under B. F. (Sales Tax) Act, as they are not considered as dealers and manufacturers of skiagrams."

THE SECOND TUBERCULOSIS SEAL SALE CAMPAIGN

"In the matter of T.B., India is on the verge of a precipice. The disease kills five lakhs of people every year and leaves another 25 lakhs as active cases. These are indeed staggering figures and it is still more frightening to know that far from being confined to our large cities the disease is spreading to small towns and even to the sparsely populated villages. It is therefore our duty to assist agencies engaged in developing measures to alleviate the sufferings of these unfortunates and to prevent the further spread of this scourge. The T.B. Seal Campaign is one of the methods directed to achieve this end.

"Nothing will be of greater service to the nation than that every man and every woman in this country should take to the T.B. Seal as a national duty calculated to convey the cheering message that we are not going to forsake our brethren who are in distress.

"I appeal to the People of India to co-operate with their T.B. Associations and look upon the T.B. Seal as a symbol

of peace, cheer and consolation. Much, indeed everything, can be attained with the people's goodwill and co-operation. Jai Hind."

The above are the excerpts from the observations by the Hon'ble Rajkumari Amrit Kaur, Minister of Health, in a broadcast talk delivered from All-India Radio, Delhi, at 9-45 P.M. on October 1, 1951.

* * * * * *

The Second Seal Sale Campaign was inaugurated in West Bengal by Dr. K. N. Katju on October 2, 1951. The Campaign will come to an end on 26th January, 1952. The collections from the First Campaign in West Bengal in 1950-51 was Rs. 1,12,128-15-6 (up to 25th October '51). In the current campaign, the Committee of the Bengal Tuberculosis Association hopes to raise a much higher figure and thus to extend its anti-T.B. activities more vigorously and extensively. The Committee expects all physicians to do their part in this nation-wide campaign against T.B.

THE FOURTH INDIAN PHARMACEUTICAL CONGRESS, JAIPUR

Arrangements are being made for the fourth session of the Indian Pharmaceutical Congress to meet in Jaipur during the end of December 1951. Shri S. P. Sen, will preside. Col. M. K. Kelavkar, Director of Medical and Health Services, Rajasthan, will welcome the guests as the Chairman. The programme will include science session for the reading and discussion of research papers.

CALCUTTA MEDICAL CLUB

Golden Jubilee Celebrations

The Golden Jubilee Celebrations of the Calcutta Medical Club will be held in the club buildings at 91/B, Chittaranjan Avenue, Calcutta 12, from the 26th December 1951 to the 15th January 1952. The programme will consist of scientific lectures by eminent scientists and medical men, scientific exhibitions, visits to medical, pharmaceutical and scientific institutions and social functions. Three prizes, of the value of Rs. 250/- each, will be awarded to the authors of three best articles on purely medical subjects.

CORRESPONDENCE

The Editor is not responsible for any views expressed by contributors

RICE DIET & FERTILITY CONTROL—BY CONTRA-FERTILISER OF MASS POPULATION PLANNING BY STATE

Sir,—Some time back, leading local papers gave prominence to the caption—" Rice Eating and Fertility. Poser for New Delhi " and went on as follows: " Is rice-eating responsible for increased Fertility, leading to greater pressure on rice and millets in India? *This is the interesting poser before Government of India authorities* examining the recent census figures."

I would like to point out that this is not a new discovery. Japanese experts have worked on the subject. In 1934, at the G. S. Medical College, a meeting of the medical profession was held to discuss 'birth control.' During the discussion, I pointed out that to check the excessive growth of population in India we must understand the underlying cause of such increased fertility, which was the rice dietary of our people. That one of the radical methods of fertility control was to control the rice diet. In support thereof, I alluded to the observations, experiments and research work of certain renowned Japanese scientists. They have brought forward convincing evidence that rice contained definite amounts of " fertility factors," which they succeeded in isolating. These results were published in scientific as well as lay journals.

From the scientific point of view, the high human fertility of rice diet can be countered and eliminated in two different ways, *viz.*, one, by the " prohibition " of the cultivation, import and eating of rice which is not a practical proposal, looking at the age-long dietary habits of our people. The alternative is the method of scientific research. Modern science has advanced so far as to create new species and genera of animals and plants. So, research can create a new variety of rice, by the elimination of the " Fertility Factors " from the new rice, and so, " sterilise " the new rice and radically solve the problem of excessive fertility of our people.

Or otherwise, scientific research can create some new substances that when added to rice even in small quantities will be capable of destroying the " Fertility Factors " in rice safely and without any harm to the nutritive qualities of rice. Such " Anti-Fertility Substances " *i.e.*, Contra-Fertilisers must be cheap, tasteless and easily available in the market. The public must be instructed in the need and value of " Fertility Control " through such means to reduce the appalling poverty and misery of our teeming millions of half-starved men and women.

My submission is that the Central and State Governments in India must endow new fertility research institutes in India, with well-defined objectives, *viz.*, investigate into the " Fertility Factors " in the varying dietaries of the people within their jurisdiction, and the methods of combating same, (as suggested above), and also the problems of sterility in human and animal beings, dependent on their varying diets. This is a vital and grave problem for India, with its over-population and its annual increase of 2 per cent per annum. So far as one knows, such institutes do not exist anywhere, but that is no reason why India should not be the pioneer in this vital field of research.

Such institutes must have other objectives as well, *e.g.*, they can investigate into the metereological factors affecting fertility, and also the differential fertility of the sexes, dependent upon the temperatures, humidity, etc., of the various parts of India, which would explain why there are more girls than boys (or *vice versa*) born under these varying factors. Obstetricians of long experience have described in the Reports of their Maternity Hospitals, etc., that more girls are born in humid maritime climate as in Bombay, and more boys in dry climates. That will explain the disparity in numbers of sexes in different parts of India, and enable parents to have the choice of sex determination. There are other relevant problems, which can be solved by the proposed fertility institutes, such as of soil fertility.

The distinction between Birth Control and Fertility Control is clear. If there were efficient exercise of " fertility control," there will be no need for birth control, as excessive births due to rice diet will be prevented. Only if the fertility control fails, birth control will be needed.

Before the advent of research into the fertility factors in rice, birth control clinics are bound to be expensive, and provision for 35 crores of our population, plus annual increase of 2 per cent would require many lakhs of such clinics and heavy expenditure thereon. Hence, one advocates research in fertility control, that would prove cheaper and of universal application through changes in dietary of our people.

Again, objections on so-called moral or religious grounds to birth control will be automatically eliminated by the new approach and sure preventive check to over-population through fertility control, which will, in fact, prevent unwanted births and the accompanying untold human misery. Rice is the most practical and cheapest human fertiliser, and research as proposed above can sterilise it by removing the " Fertility Factors " in its composition, by contra-fertilisers.

Vital Statistics of our population bear eloquent testimony to the terrible but preventible wastage of child life. High fertility implies high child and maternal mortality in India. Out of every 100 deaths in India, 25 die in the 1st year of life—(Infant Mortality), plus 25 die in the first 10 years of life, *i.e.*, half the total deaths are among children under 10 years of age! Child Mortality—Do Indian mothers breed unwanted children that they die within the first 10 years of life? And need 20 overburdened mothers die per 1,000 live-births? How much wealth of India is drained away by the funeral and post-funeral expenses, and at what cost to the health and lives of mothers? Where is the moral or religious merit in this holocaust? Fertility control can and will prevent this endless misery of our masses.

Again, the expectation of life for Indians is the lowest of any other people in the world—*viz.*, 25 years as computed but 21 years in fact.

Till to-day, population commissions, conferences etc., have been held all over the world, and reports, books and pamphlets unnumbered have been published on the subjects of family and population planning, emphasising the benefits of birth control, but these are inapplicable to poor India. Films on planned parenthood are also shown with very poor results.

The other day, the local Minister of Health is reported to have said that " the time has not come yet for direct intervention of the State in this problem of over-population." And that " the principle of family planning was accepted by the Planning Commission of India, and members differed only on the methods to attain the goal." The main submission of this article is that the time is ripe for fertility control by the State as suggested herein, and that the Planning Commission need set apart the essential finances for fertility research institutes in India, to carry out the objectives outlined above. Instead of the expensive and tedious family planning or birth control by individuals, the Government can exercise mass planning of population through fertility control—the most effective approach to this problem. It may be added that in cases where primary fertility control fails, secondary birth control can be utilised. In the age of Malthus, science had not advanced to the stage it has reached in our days, when we can deal scientifically with the problem of over-population and solve it by the control of fertility factors in rice in particular and in other dietary articles in general. I am etc.

13, Jenkins House,
Merewether Road,
Bombay. K. K. DADACHANJI

I.M.A. REPRESENTATIVE ON BOARD OF FILM CENSOR

Sir,—Not infrequently in the lay press and on the screen incidents are portrayed which are positively defaming the medical profession. Of the two, the screen, is more powerful and hence I suggest that a doctor representative of our Association should be on the censor board or at least one should be invited to witness the film so that he may bring to the attention of the censors in time any defaming scenes if present before the film is released.

I cite a concrete example. In the Telugu language film " Samsaram " a doctor refuses to attend on a patient unless his fees are paid in advance. This starts a chain reaction leading to violence. Heads are broken and the doctor nearly gets choked off by the patient's impatient and loving but poor wife. Ultimately, the doctor relents and makes ample amends for his past behaviour.

The uncultured audience naturally fails to grasp the doctor's noble gesture, only his early " villainy " sticks in their memory.

The uncultured audience cannot naturally discern the nobility which the doctor later displays. I definitely feel that scenes which are likely to encourage violence to and defamation of a responsible profession must necessarily be censored. I am etc.

Tekkali (Srikakulam Dist.). M. V. RAMA RAO, M.B.B.S.

REVIEWS

HANDBOOK OF MEDICAL MANAGEMENT—By Milton Chatton, A.B., M.D., Sheldon Margen, A.B., M.D., & Henry Brainerd, A.B., M.D. Second Edition, 1951. Published by University Medical Publishers, P. O. Box 760, Palo Alto, California. Sole Distributors for India, Pakistan, Burma and Ceylon: The Kothari Book Depot, King Edward Road, Parel, Bombay 12. Pp. 507. Price Rs. 14/-.

In the management of diseased states, diagnosis undoubtedly remains the most important step. Even after the correct diagnosis has been made, one at times feels the necessity of a handy volume giving all the relevant information regarding medical management in concise and well arranged form. This handbook of medical management is sure to fill such a need of medical practitioners. The subjects-matter have been well chosen and also well arranged. The informations are given in detail whenever necessary and includes even the most recent advances. This small handy treatise will be received with considerable enthusiasm by the practitioners in general and will be found to be very useful for everyday medical practice.

S. C. C.

THE VITAMIN B COMPLEX—By F. A. Robinson, M.SC., LL.B., F.R.I.C., Published by Messrs. Chapman and Hall; 37 Essex Street, London, W. C. 2, First Edition, 1951, Price 60 s. net. Pp. xi. plus 688.

This is an excellent book, covering the various fields and diverse subdivisions of the vitamin B complex and its different factors, written by one who has made a study of the subject for over fifteen years and has been engaged in the

production, isolation and assay of the most important members of the B complex. The whole subject has been extensively dealt with, neatly divided into 14 chapters each of which has received the due attention it deserves. Each chapter has been further subdivided into several sections, at the end of which there is a select list of references for those who want to pursue their studies further. The entire field of the various factors of the vitamin B complex has been ably discussed and presented to the reader in a nice and erudite way which cannot but fascinate the ardent reader. This book should find an honoured place on the table of every medical man and scientific worker interested in the complexities, importance and deficiencies of the vitamin B complex.

R. S.

DISEASE IN INFANCY AND CHILDHOOD—By Richard W. B. Ellis, O.B.E., M.A., M.D., F.R.C.P. Published by E. & S. Livingstone Ltd., 16 & 17, Teviot Place, Edinburgh, 1951. Pp. 695 plus viii. Price 42 s.

A book by Professor Ellis is always welcome as some new angle of pediatrics is sure to be focussed in it. This handbook of diseases of children is really the complement of his earlier volume "Child Health and Development" which was a great success. The aim of the book is "primarily to emphasise those features of the young and growing individuals which will determine his response to the impact of determine processes, and distinguish this response from the behaviour of the adult whose development is complete or whose organs are already suffering degeneration."

The book, therefore, is not divided into regional chapters but rather the growth and development formed the basis of these differentiation.

The first chapter is headed history-taking and examination. Much thought has been devoted in writing this chapter and inclusion of a discussion of behaviour problem in detail is a welcome addition as 'these represent an important field of ill-health in childhood.'

The next two chapters consider such factors as social and environmental factors in disease and constitutional factors in disease. The first chapter is new in pediatric literature and is a very important one towards understanding of social pediatrics.

The three following chapters consider the newborn and its diseases followed by disorders of nutrition and digestion, disorders of storage, disorder of growth and development, neoplastic diseases, disorder of blood, allergic diseases, rheumatic disorders, diseases of the gastro-urinary system, tuberculosis, non-tuberculous disease of the respiratory tract, communicable diseases and other infections, degenerative and miscellaneous disorders, behaviour disorders, procedure and therapy.

The book is replete with beautiful photographs of a number of typical cases and the get-up is upto the usual Livingstone standard.

The real difficulty of the book is its high price. One will think twice before buying a book of 42 s. specially when it is "on introduction to clinical pediatrics" only.

N. G. M.

FAMILY PLANNING—WHY? WHEN? & HOW?—By Lt.-Vol. B. L. Raina, A.M.C. Published by New Book Company Ltd., 188-190, Hornby Road, Bombay. Third Edition, 1951, Pp. 100. Price Rs. 3/12 as.

In this small and handy pocket-size book the author has made an attempt to present the different aspects of the subject of conception control, subdivided into the three sections as indicated in the title of the book. The first chapter (Why?) appears well presented with good arguments and display of comparative figures, almost all of which however are from foreign sources. The various methods of family planning do not seem to have been fairly dealt with, being too inadequate a guide for the lay public and often superfluous for the medical men. Over 40 pages out of the total of 100 pages have been devoted to appendices, and the bulk of this material seem irrelevant to the purpose of the book as outlined in the title-page. Printing and binding of the book are, on the whole, fair.

R. S.

YOUR QUESTIONS

Correspondents should give their names and addresses (not necessarily for publication) and include all relevant details in the questions which should be typed. Questions of medical interest only will be acceptable for this section. They should be sent in a separate envelope marked "Your Questions." Questions of general interest will be published in preference. Lack of space precludes answering all the questions received.

Q. 1. *What is the difference between rock salt and common salt? I have got a patient suffering from hypertension. He has been advised to use rock salt instead of common salt in his diet. Is there any justification of this advice?*

Ans. 1. There is not much difference between "Rock salt" and "Common salt" excepting that the former contains some impurities and some calcium in addition to sodium chloride which itself is "Common salt" in a purified form.

There is no rationality of prescribing rock salt in place of common salt since both of them contain NaCl, claimed to be the offending agent in causing hypertension. It is thought that NaCl particularly the Na-ion is responsible, at least to some extent, for production of hypertension. Hence, a salt-poor (so-called salt-free) diet is advocated as a preliminary to drug treatment in persons suffering from essential hypertension with some benefit. The results are not uniform. The mechanism by which B.P. is lowered on withdrawal of the Na-ion from the diet is not clear.

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Finally, a note of warning is also required in this connection, since low-sodium diet is not always without danger. In patients with impaired renal function a low-sodium diet may produce a condition called "salt-depletion syndrome" which is characterised by headache, anorexia and abdominal cramps followed by oliguria and ultimately death from uræmia.

Q. 2. "In a house where a case of diphtheria is being treated, can a cat with a swollen neck be considered as diphtheritic and cause of infection?"

Ans. 2. No, the case against the cat has never been satisfactorily proved.

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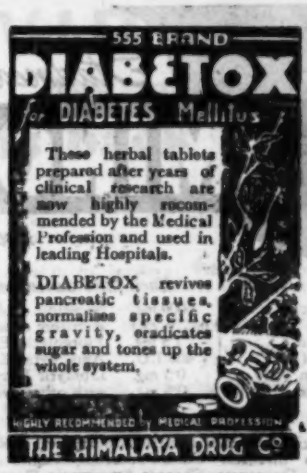
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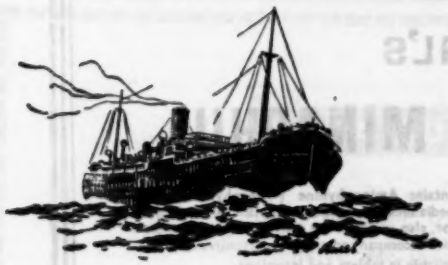

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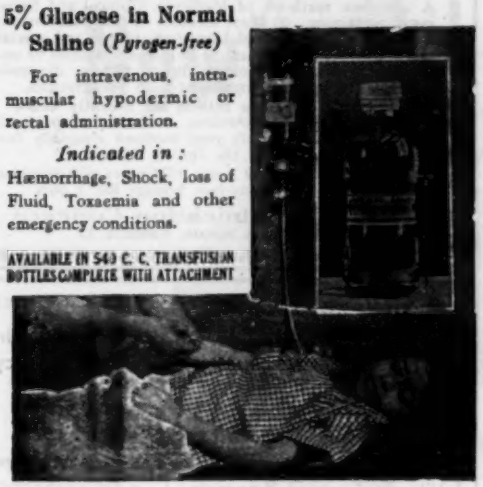

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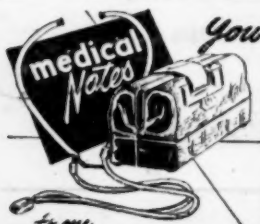

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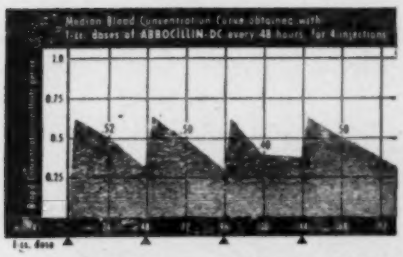

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